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Abstract

Title of Thesis: The Effects of Endotracheal Suctioning with Manual Hyperventilation on Cerebrovascular Status of Closed Head Injured Patients.

The main goals of nursing care of the head injured patient include maintenance of normal intracranial pressure (ICP) and prevention of complications of immobility. Pulmonary complications of immobility are prevented by regular endotracheal suctioning for intubated patients. The purpose of this study was to determine the effects of endotracheal tube suctioning (ETTS) with manual hyperventilation (MH) on the cerebrovascular status of severe closed head injured patients. In contrast to previous studies of endotracheal suctioning, the frequency of measurement and treatment protocols were designed to more accurately reflect usual clinical situations.

A convenience sample of 20 mechanically ventilated patients with closed head trauma, a mean arterial blood pressure (MABP) > 50mm Hg, and PaO2 > 70mm Hg, were studied utilizing a within-subject repeated measure design. ICP was

monitored via subarachnoid bolt or fiber-optic catheter. All had been admitted to the neurotrauma center or critical care recovery unit at a large metropolitan trauma center. Subjects were continuously monitored during the protocol using a four-channel recorder to quantify ICP, arterial blood pressure, heart rate (HR), and oxygen saturation (O2 SAT) levels and provide a time dimension over which to assess the resultant values. The dependent variables: ICP, MABP, HR, and O2 SAT were recorded 5 minutes prior to and immediately following the 3 minute ETTS/MH protocol. All data were collected by a 2 person nursing research team utilizing a standardized protocol.

Repeated measures Analysis of Variance (ANOVA) was computed for data analysis. The research protocol closely replicates actual clinical practice techniques and illustrated a significant rise ($p = .000$) in ICP during the ETTS/MH phase with a return to within 1 mm Hg of pre-treatment levels during the 5 minutes post-procedure. Cerebral perfusion pressure (CPP) values stayed above 70 mm Hg throughout the procedure which is well above 50 mm Hg, a minimal value needed to insure adequate cerebral blood flow. Effects of transient changes on long-term recovery of head injured patients is not clear. Further research regarding long-term effects of transient changes as well as refinement of nursing practice to minimize these changes is indicated.

The Effects of Endotracheal Suctioning with Manual Hyperventilation on Cerebrovascular Status of Closed Head Injured Patients

Darnell M. Waun

Thesis submitted to the Faculty of the Graduate School of the University of Maryland in partial fulfillment of the requirements for the degree of Master of Science
1989

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DEDICATION

Dedicated to my wife Jolene and
my children, Michael and Andrea
for their unwavering support, patience,
and encouragement during the completion
of this thesis.

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The author wishes to express his gratitude to Dr. Sue Thomas, Dr. Karen Soeken, Kathryn Von Rueden, and Connie Walleck for their unwavering support and guidance throughout this thesis. Their support was invaluable in the development, implementation, and completion of this study.

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A special thanks goes to Chip Galloway who assisted in preparation of the graphic representation of the data.

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Military Disclaimer

The author is a member of the United States Air Force Nurse Corps. The opinions and assertions contained herein are those of the author and are not to be construed as reflecting the views of the Department of the Air Force or the Department of Defense.

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CHAPTER I

INTRODUCTION

Trauma is the leading cause of death of persons in the first three decades of life in the United States and the number three killer overall behind cancer and heart disease (Caveness, 1979). Brain injury accounts for at least 50% of all trauma deaths (Trunkey, 1985). It is estimated that over one million people per year suffer major head injuries in the United States (Caveness, 1979). Individuals with severe head trauma present significant medical and nursing care challenges. A major goal of the care of these individuals is the maintenance of adequate perfusion of the brain. Serious questions as to the effects of routine nursing care procedures such as endotracheal tube suctioning on brain perfusion, have been raised. A study by Mitchell and Mauss (1978) demonstrated a rise in intracranial pressure (ICP) during routine care activities such as turning and suctioning. Increased ICP is the leading cause of death following severe head injury and the primary cause of morbidity in head injury survivors (McGinnis, 1988).

Pulmonary infection ranks second only to intracranial hypertension as the leading cause of head injury death (McGinnis, 1988, p. 395). A second therapeutic goal for head injured patients is prevention of pulmonary

complications utilizing optimal pulmonary toilet and frequent suctioning of intubated patients. Studies indicate that endotracheal suctioning causes hypoxemia and hypercarbia which increases cerebral blood volume and an increase in ICP (Fisher, Frewen, & Swedlow, 1982; Parsons & Shogan, 1984; Shalit & Umansky, 1977; Snyder, 1983; Tsementzis, Harris, & Loizow, 1982).

Theoretical Framework

Within the bony confines of the skull is the intracranial cavity. Three components comprise the volume of this cavity: brain tissue (80%), blood (10%), and cerebrospinal fluid (CSF) (10%). Intracranial volume is fixed externally by the rigid skull, which restricts expansion of the intracranial volume. The Monro-Kellie doctrine or "box theory" states that an increase in one volume component must be offset by a reciprocal decrease in one or both of the remaining components to maintain a constant volume within the cranial cavity (McGinnis, 1988). In the presence of an intracranial lesion such as a tumor or edema, CSF will initially be displaced into the distensible spinal subarachnoid space, followed by CSF absorption as ICP increases, with a reduction in intracranial blood volume if further spatial compensation is required (Shapiro, 1975). Eventually, the intracranial capacity to compensate via CSF translocation will be overcome, brain elastance will diminish and ICP will rise rapidly (Mendlow & Teasdale,

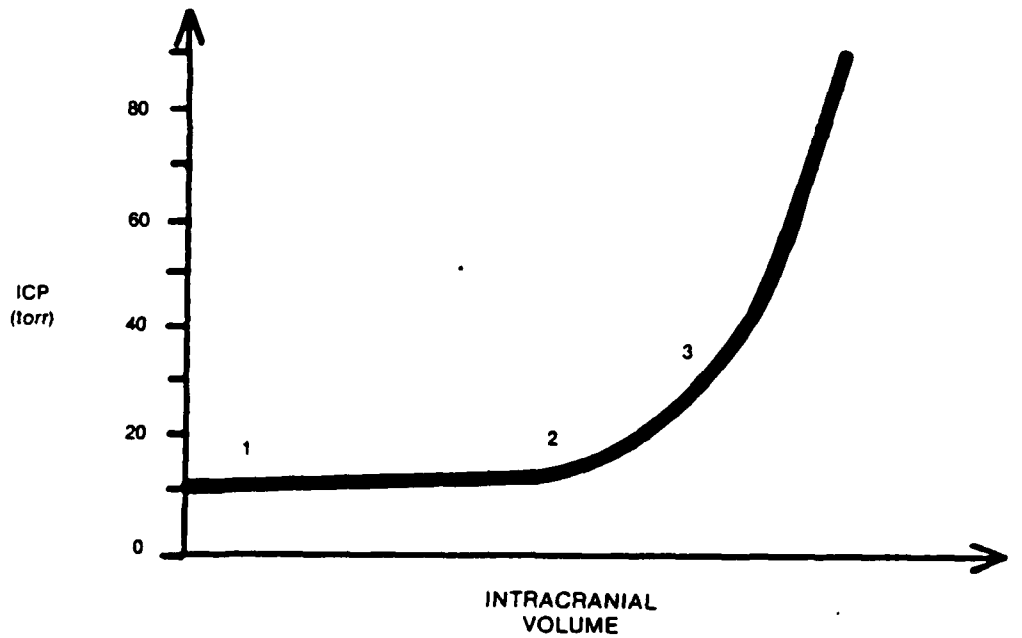
1983). Sustained intracranial hypertension causes a reduction in cerebral blood flow (CBF) needed to maintain cerebral metabolism (Langfitt, 1976, McGinnis, 1988).

Normal ICP ranges from 0 to 10 mm Hg. In patients with head trauma, mortality approaches 65% when the ICP rises to 40 mm Hg. When ICP increases further, to over 60 mm Hg, mortality is invariably 100% (Miller, Becker, Ward, Sullivan, Adams, Rosner, 1977). The relationship between elevated ICP and morbidity is not well developed. Reducing moderately elevated ICP (25-40 mm Hg) does not ensure a good clinical outcome devoid of irreversible neurologic deficits (McGillicudy, 1985).

Compliance, or the ratio of the change in volume to the resulting change in pressure, is applicable to intracranial dynamics. Figure 1 demonstrates the relationship between the volume of the intracranial compartment and ICP (Shapiro, 1975). With normal intracranial volumes (point 1), the ICP is low and remains so even with added volume. This is the result of adequate compensatory mechanisms and high compliance. As volume increases (point 2) , compensatory mechanisms are exceeded, and further addition of volume results in greater rise in pressure. When ICP is elevated, additional volume expansion results in marked increases in ICP (point 3).

Figure 1

Volume-Pressure Curve



(From: Shapiro, H.M. (1975). Anesthesiology, 43, p. 447)

The normal brain receives a total of 750 ml of blood per minute or 50ml/100gm/minute. The human brain is 2% of total body weight, receives 15% of the cardiac output, and accounts for 20% of total body oxygen consumption. This disproportionate amount of flow is necessitated by the brain's high metabolic rate and oxygen requirements (Chien, 1985). The CBF is maintained by cerebral perfusion pressure (CPP), which is equal to the difference between systemic arterial pressure and ICP (Gaab & Heissler, 1984). The normal range for CPP is 50 to 130 mm Hg with an average of 80 to 100 mm Hg. Values less than 50mm Hg are indicative of decreased perfusion and ischemia; values greater than 130 mm

Hg are associated with hyperemia, that is blood flow in excess of metabolic demands of the tissue (Langfitt & Obrist, 1981; McGinnis, 1988).

Various mechanisms impact brain autoregulation. Pressure or myogenic autoregulation is the brain's ability to maintain a constant rate of blood flow over a wide range of perfusion pressures despite changes in arterial blood pressure and ICP (Mendlow & Teasdale, 1983; Gaab & Heissler, 1984; McGinnis, 1988). Cerebral arterioles, the resistance vessels, are the primary agents of pressure regulation because of their ability to independently dilate or constrict in response to metabolic and pressure variations. These vessels have an inherent "self-regulatory" mechanism, which allows them to respond to changes in transmural pressure. As a result, an increase in arterial pressure causes vasoconstriction of the vessels, and a decrease in arterial pressure causes vasodilation. This mechanism maintains CBF essentially constant for a range of mean arterial pressures between 50 to 170 mm Hg (McGinnis, 1988).

Metabolic factors have a marked effect on CBF. These include PaO2, pH, and PaCO2. A decrease in PaO2 below 50 mm Hg (hypoxemia) stimulates cerebral vasodilation and increases CBF in adults (Rockoff & Kennedy, 1983; McGinnis 1988). The resultant increases in cerebral blood volume as PaO2 falls causes large elevations in ICP when intracranial compliance is low. Extracellular reduction in pH, cerebral

acidosis, caused by hypercarbia or lactic acid accumulation in ischemic brain tissue, causes vascular dilation and increases CBF and volume (McGinnis, 1988). Carbon dioxide (CO2) is the most potent vasodilator of cerebral vessels, and a sudden change in PaCO2 normally changes CBF 2 to 6% for every mm Hg change in PaCO2 resulting in an upward shift in cerebral volume (Enevoldsen, 1986). This mechanism is the underlying principle for hyperventilation, the primary treatment modality for increased ICP. Hyperventilation aids CO2 removal and results in constriction of cerebral vessels, reducing cerebral volume and decreasing ICP (Langfitt, 1974; Rockoff & Kennedy, 1983).

When autoregulation is intact, CBF is maintained during rising ICP (Langfitt, 1969). Autoregulation can be determined to be intact if CPP remains within the normal range (50-130 mm Hg) and there are no rises in ICP accompanying increases in mean arterial blood pressure (MABP). If the rise in ICP does not cause the limits of CPP to be exceeded, then the cerebrovasculature should be able to regulate itself to maintain a constant, adequate CBF. Once outside this range, CBF becomes passively dependent on the CPP and the delivery of metabolites is impaired. When MABP falls below 50 mm Hg, a reduction in CBF and perfusion occurs, resulting in ischemia. Likewise, surpassing the upper limits (170 mm Hg) impairs the brain's autoregulatory mechanism, signifying that reactivity and responsivity of

the resistance vessels is lost with disruption of the blood-brain barrier, followed by passive dilation and increased flow (Larsen & Lassen, 1979; McGinnis, 1988). With loss of autoregulation, CBF rises and falls according to the CPP (Mendlow & Teasdale, 1983).

In summary, the normal brain maintains a system of regulation that matches cerebral blood supply to metabolic demand. The cerebral blood volume varies inversely with changes in cerebrovascular resistance. Injured brain tissue may not be able to compensate for increases in intracranial volume that occur secondary to changes in intracranial blood volume or the injury itself. Autoregulation may be lost globally or locally in an unpredictable and unquantifiable manner (Rockoff & Kennedy, 1983). The goal of therapeutic decision making must be to prevent significant and sustained increases in ICP to minimize destructive consequences (Shpritz, 1983).

Statement of Purpose

The purpose of this study is to increase the body of knowledge on the effect of nursing activities on head injured patients. This study examines the effects of endotracheal suctioning with manual hyperventilation (ETTS/MH) on the cerebrovascular status of severe closed head injured patients utilizing a standardized protocol.

Research Question

What is the effect of the ETTS/MH procedure on the cerebrovascular status of patients with severe closed head injury as measured by changes in mean arterial blood pressure (MABP), mean intracranial pressure (MICP), cerebral perfusion pressure (CPP), heart rate (HR), and oxygen saturation (O2 SAT)?

Definitions

Intracranial Pressure - the force exerted against the resistance of the skull by the cranial contents which includes brain tissue, blood, and cerebrospinal fluid as measured by an intraventricular catheter or subarachnoid screw.

Mean Arterial Blood Pressure - an average of the tension of the blood within the arteries. Equivalent to one-third the pulse pressure (systolic minus diastolic) added to the diastolic blood pressure.

Cerebral Perfusion Pressure - the force available to cause blood to flow through the vascular bed of the brain. CPP is equivalent to the difference in intravascular pressure between the arteries that enter the craniospinal cavity and the veins leaving it. Its value can be closely approximated by calculating the difference between the mean arterial blood pressure and intracranial pressure.

Heart Rate - the number of times per minute the heart contracts.

Manual Hyperventilation - delivery of inflations at increased rate and oxygen concentration to raise alveolar minute ventilation through use of a manual ventilation bag.

Closed Head Injury - mechanical blow to the head resulting in cerebral trauma demonstrated by decreased level of consciousness without penetration of the dura mater.

Rest Periods - Time frame in which no therapeutic interventions or physical contact with the patient is occurring.

Assumptions

1. Autoregulation is intact.

2. Environmental conditions such as noise, temperature, and lighting will not affect ICP.

Significance of Study

Planning and implementation of nursing care for the head injured patient requires that the nurse be aware of the effects of patient care activities on the cerebrovascular status of the patient. Prevention of pulmonary complications of immobility necessitates regular endotracheal suctioning for intubated patients. If the ultimate goal of the care of these individuals is maintenance of adequate brain perfusion, the effects of

current suctioning protocols need to be studied to determine how this procedure affects the patient's intracranial pressure and cerebral perfusion.

To date, studies related to intracranial pressure have focused on the pathophysiology and treatment of the problem of increased intracranial pressure. Studies by Parsons, Vollmer, Crosby, and Holley-Wilcox (1983) and Parsons and Shogan (1984) specifically address nursing care measures, including suctioning, in an attempt to identify nursing interventions which lower or prevent significant sustained increases in intracranial pressure.

The purpose of this study was to determine the harmful or beneficial effects of endotracheal suctioning with manual hyperventilation on the cerebrovascular status of severe closed head injured patients. In contrast to previous studies of endotracheal suctioning, frequency of measurement and the treatment protocols were designed to more accurately reflect clinically relevant situations and thus, to reveal clinically important variations in cerebrovascular status. Findings from this study will influence the care delivered by nurses and other health care professionals to this patient population.

Organization of the Thesis

Chapter I introduced the reader to the problem studied by the researcher.

Chapter II presents the review of literature related to the study. Related studies on suctioning, intracranial pressure, and the effects of nursing interventions is presented.

Chapter III focuses on the methodology utilized in this study. Design, subjects, data collection, instrumentation, protection of human rights, and procedures for data analysis are discussed. Limitations of the study are identified.

Chapter IV presents the results of this study.

Chapter V presents the discussion of results, conclusions, implications for nursing practice, and recommendations for further study.

CHAPTER II

Review of Related Literature

Nursing care activities have been shown to cause significant increases in intracranial pressure (ICP). As a result, intracranial hypertension has become a phenomenon of central interest for neuroscience nursing research. The majority of this research has been descriptive of the influence of various nursing care activities on ICP, namely, establishing an understanding of aspects of nursing care and patient activity that may act as demands upon the intracranial system.

This chapter presents literature relevant to the focus of this study which is the effects of suctioning and manual hyperventilation on the cerebrovascular status of closed head injured patients. Literature related to the measurement of intracranial pressure and cardiovascular response to suctioning is included. Research conducted under the broad umbrella of "nursing activities" and their effect on cerebrovascular status with designs that address suctioning and/or hyperventilation effects is also reviewed. Finally, studies specifically addressing suctioning and its effect on cerebrovascular status are discussed.

Intracranial Pressure Monitoring

Continuous monitoring of ICP provides a wealth of information concerning the diagnosis, treatment, and prognosis of patients with sustained or intermittent

elevations in intracranial pressure (Hanlon, 1976). Until technology develops to the point that cerebral blood flow can be measured directly, prevention of abnormal metabolic demands and maintenance of adequate cerebral perfusion pressure (CPP) must be computed using intracranial pressure and other physiological measurements such as mean arterial blood pressure. Elevations in ICP that can compromise CPP are not necessarily apparent on clinical examination (change in level of consciousness, change in pupil equality and reactivity, widening pulse pressure and bradycardia) (Rockoff & Kennedy, 1983). When these signs are associated with increased ICP, they are usually late and dangerous symptoms. ICP monitoring permits detection of altered ICP at an early stage and permits observation of trends over time.

Lundberg (1960) described ICP and monitoring in brain-injured patients employing direct cannulation of the ventricle. The intraventricular catheter (IVC) is still considered the optimal way to monitor ICP (Walleck, 1987). Advantages of this system include reliable measurement within cerebral spinal fluid (CSF) which also gives access for CSF drainage and sampling and for determination of volume-pressure response. Disadvantages include difficulty locating the lateral ventricle and risk of intracerebral bleeding or edema at the cannula track and infection (Hollingsworth-Fridlund, Vos, Dailey, 1988).

The subdural screw is currently the device most widely used for continuous ICP measurement. This is due primarily to the relative ease of insertion and less invasive nature compared to the intraventricular catheter. Its use is advantageous if the ventricles are small and the infection risk is considered minimized because the site of infection is frequently limited to the scalp (Hollingsworth-Fridlund, et al, 1988). The major disadvantages are that CSF cannot be drained through this device and pressure readings can be unreliable when high ICP herniates brain tissue into the bolt.

Mendelow, Rowan, Murray, and Kerr, (1983) compared multiple recordings from the single lumen screw (Richmond type) with simultaneous recordings obtained by ventricular catheters in ten patients. They found that when ventricular fluid pressure exceeded 20 mm Hg, there was an increasing tendency for the subdural screw pressure readings to be lower that those obtained with the intraventricular catheter. However, when pressures ranged between 0 to 10 mm Hg, the screw pressures corresponded to the ventricular measurements 86% of the time.

Epidural sensors and fiber-optic transducer tipped catheters have been introduced as the latest method for measurement of ICP. The key to the system is fiber-optic transmission of pressure from a miniature transducer at the tip of the catheter. Inside the transducer, a mirrored

diaphragm moves in response to pressure and is sensed by the light fibers. Its major advantages are ease of insertion, no dural penetration, lower risk of infection, and no adjustment of transducer position is needed. Its disadvantages include inability to drain CSF or recalibrate or rezero after placement. The accuracy of sensing ICP through the dura is still under investigation (Hollingsworth-Fridlund, et al, 1988).

Effects of Suctioning on Cardiovascular Status

McQuillan (1986) studied a convenience sample of 20 mechanically ventilated hemodynamically and respiratory stable adult subjects with a diagnosis of open or closed head injury to determine the acute physiologic alterations in cerebrovascular status that occurred during chest physiotherapy (CPT). ICP was measured by subarachnoid bolts or intraventricular catheters and MABP by arterial line. Cerebral autoregulatory mechanisms were determined to be intact. The range of baseline ICP's was not specified. MABP and HR were used to determine the cardiovascular effects during the protocol which included suctioning. Suctioning is defined as passing a suction catheter into the endotracheal tube, applying suction for no more than ten seconds, and withdrawing. The patient is ventilated with 100 percent oxygen by ambu bag for 20-30 seconds before and after suctioning.

ICP, MABP, and HR all rose during the treatment period and declined various degrees following the treatment, however none fell below baseline levels. The rationale cited was that ICP elevations stimulate the cerebral ischemic response subsequently activating the sympathetic nerves to increase HR and MABP. A strong positive correlation ($p < .001$) between CPP and MABP was identified throughout the CPT procedure. The findings supported the importance of maintaining a sufficient MABP in assuring adequate perfusion of blood to the brain. A limitation of the study is the fact that the suctioning procedure was done after 10 minutes of vigorous stimulation from chest percussion and vibration which further elevated arousal and release of catecholamines into the system. This could have resulted in delayed clearance of the catecholamines which would account for the MABP staying above baseline following the procedure. The results of this study are only generalizable to the hemodynamically and pulmonary stable head injured population.

Winston, Gravelyn, and Sitrin (1987) used a convenience sample of six adults to compare the effectiveness of nebulized atropine (0.05 mg/kg ideal body weight) with that of parenterally administered atropine (1 mg IM or slow IV push) in preventing bradycardia and hypotension related to endotracheal suctioning. All subjects were intubated and required mechanical ventilation with maintenance of adequate

oxygenation (PaO2 > 60torr). Patients had acute pulmonary conditions including pneumonia, adult respiratory distress syndrome, or sepsis. HR was monitored continuously via a bedside cardiac monitor and systolic BP was measured with a sphygmomanometer at baseline, immediately after suctioning, and every 15 minutes for one hour after suctioning. The suctioning procedure was not specified. All subjects were removed from the ventilator and pre-oxygenated with 100% oxygen by manual bagging for an unspecified period of time.

All six patients experienced a fall in resting HR with suctioning after nebulized saline. Mean HR fell from a baseline value of 114 +/- 10 to 45 +/- 5 beats/min ($p < .001$). HR fell within seconds of endotracheal suctioning and returned to baseline within 15 minutes. Both parenteral and nebulized atropine given 30 minutes prior to suctioning prevented bradycardia in all six patients. Three patients experienced a fall in systolic BP with endotracheal suctioning. Mean systolic BP with suctioning fell from a baseline of 128 +/- 8 to 87 +/- 24 mm Hg after the nebulized saline trial. Both nebulized and parenteral atropine sulfate completely prevented the hypotension related to suctioning.

There are many limitations in this study. The first was the small sample size. The suctioning procedure was not specified which limits generalizability. Acute changes in BP cannot be sensed using a sphygmomanometer and taking

measurements every 15 minutes which raises questions regarding the sensitivity of the data to the actual response of the subjects. Inhibition of bradycardia and hypotension elicited during endotracheal suctioning by atropine sulfate suggests that these responses are at least partly vagally mediated.

Rogge, Bunde, and Baun (1989) designed a within-subject study which compared the effects of hyperinflation with 20% above maintenance oxygen (O2) level and 100% O2 hyperinflation before and after endotracheal suctioning in 11 acutely ill, mechanically ventilated adult patients with chronic obstructive pulmonary disease (COPD). Blood pressure was measured using an external blood pressure monitor and an electrocardiograph machine was used for monitoring heart rate and rhythm. The suctioning procedure was the same for all subjects and included four hyperinflation and three suction episodes. The subject was given four hyperinflation breaths within 20 seconds of suctioning, a total of 15 seconds was allowed for suctioning, followed by four post-suction hyperinflations. This sequence was repeated twice.

Heart rate increased to levels greater than 100 beats/min during the second period of suctioning in both protocols with a gradual return to baseline 5 minutes after return to the ventilator. Tachycardia was attributed to anxiety or stress that the subject experienced in response

to suctioning, or coughing and resisting hyperinflation, rather than to any change in oxygen saturations.

Peak systolic blood pressure occurred at the beginning of each protocol, returning to near baseline after the patients were returned to the ventilator. Actual values were not reported. Diastolic BP had the greatest percent change from baseline. Because the greatest increase was seen at the beginning of each protocol, the initial reaction to suctioning may be a factor.

The small sample size is a limitation of this study. Generalizability of the results to the head injured population is limited due to the health status of the COPD patient population studied. The use of arterial lines for blood pressure measurement would have yielded more reliable data.

Effects of Nursing Activities on Cerebrovascular Status

Mitchell and Mauss (1978) were the first nurses to systematically study the effect of nursing activities on ICP when they conducted a descriptive study to identify which, if any, nursing activities affected ICP. They studied nine adult patients with pressure controlled ventriculostomy fluid drainage systems in place. The patients were monitored continuously for up to 24 hours to determine the incidence and amount of ventricular fluid drainage during specific activities. Incidence of ventricular fluid drainage was taken as an index of increased ICP.

Ventricular fluid drainage was associated with turning in bed, conversation about the patient, coughing, chewing, use of bedpan, and restless movements. The magnitude of change during these activities could not be demonstrated using this monitoring system. No measurements of cerebral perfusion pressure or mean arterial blood pressure were obtained which are essential for the evaluation of cerebrovascular status.

Snyder (1983) conducted a descriptive and observational study of nine adult patients with cranial pathology (7 of the 9 subjects had suffered cranial trauma) and Richmond bolts in place for monitoring intracranial pressure. The highest ICP observed during various nursing activities was recorded. Findings confirmed that nursing activities and environmental factors are associated with pressure changes. Respiratory care activities and repositioning resulted in the highest increases in intracranial pressure with mean increases above baseline of 12.7 mm Hg and 12.6 mm Hg respectively. Baseline ICP measurements were not reported. Respiratory care activities were described only as "suctioning and respiratory hygiene" and the results were confounded by other activities taking place simultaneously during the measured activities. Mean arterial pressure and cerebral perfusion pressure were not measured.

In 1981, Bruya reported the effects of planned rest periods on intracranial pressure in a sample of twenty adults who had sustained various degrees of head trauma.

Technology utilized to measure ICP as well as baseline measurements were not reported. Patients were assigned to a control group (no rest periods) or to an experimental group (planned rest periods) during the morning care routine. Nursing care activities included vital signs, hyperventilation, suctioning, oral care, bathing and bed changes. A specific standardized protocol utilized for each nursing activity was not specified. The experimental group received three 10 minute rest periods interposed between activities while the control group had all treatments done in one block of time. Rest periods were defined as leaving the patient alone in a closed room with lights dimmed. The highest ICP reading noted during each nursing activity was documented and a mean computed over all subjects. No significant differences were found between the two groups. The largest increases in ICP were found in both groups with hyperventilation, suctioning and turning. Mean arterial blood pressure and cerebral perfusion pressure were not reported. Generalizability is extremely limited because so little demographic data outside of age and sex of subjects is reported.

Boortz-Marx (1985) conducted a descriptive study observing four adult patients with either a subarachnoid bolt or intraventricular catheter in place. Three patients had closed head trauma and one sustained a spontaneous subarachnoid hemorrhage. The patients all had a Glasgow

Coma Scale score below five. Subjects were observed on three separate occasions during which the activities being measured were at their highest levels of intensity. Total observation time was 2 hours and 40 minutes. A Mennen medical chart recorder was used to provide simultaneous, permanent recordings of ICP during the study. Baseline intracranial pressure measurements were not reported. The results revealed that turning the patient, physical assessment (auscultation, palpation, pupil checks, and cuff blood pressure checks), suctioning, and nursing care measures such as bathing elevated ICP. The maximum observed elevation occurred consistently in all four patients with suctioning and resultant coughing, with a mean elevation of 10.65 mm Hg above baseline. Suctioning methodology is not noted. Minimal elevations occurred with nursing activities such as dressing changes, rectal temperatures, oral care, and drawing blood from an arterial line. Results obtained could be confounded by factors such as cumulative elevation of ICP secondary to simultaneous activities (Mitchell, Mauss, Lipe & Ozuna, 1980) and intensity of patient activities. The duration of ICP elevations and changes in mean arterial blood pressure or cerebral perfusion pressure were not indicated. These are essential in determining cerebrovascular status during the observed activities. Generalizability of the results are limited by the small sample size.

Tsementzis, Harris and Loizou (1982) in a descriptive study conducted in Great Britain, measured the effect of routine nursing procedures on ICP in patients with severe head injuries. The demographics of the sample were not identified except that there were 39 patients with a mix of adults and children. ICP was measured via intraventricular catheters. All subjects were mechanically ventilated and receiving pancuronium bromide (Pavulon). Baseline ICP's were reported to vary from 6 to 38 mm Hg. Patients were divided into two groups on the basis of the level of intracranial pressure and its response to treatment by pharmaceutical relaxants (Pavulon). Fourteen of the 33 cases in the group who had ICP controlled by relaxants displayed only a minimal reversible increase in ICP during endotracheal suctioning (range of means 1.4 to 6.5 mm Hg). Five of the six subjects in the group who were uncontrolled by use of muscle relaxants displayed a mean net increase of 35.40 mm Hg in ICP during suctioning. These rises were noted in a sample with extremely large variability in baseline ICP. They discovered no apparent relationship between resting level ICP and increase among different patients using a two-factor analysis of variance (patients vs. stimuli). The findings are difficult to interpret since no baseline measurements of pertinent physiologic parameters or quantification of the magnitude of pressure changes with each activity are documented. Blood pressure was measured

in only 22 cases and specific results are not reported. The lack of mean arterial blood pressure and cerebral perfusion pressure measurements made critical evaluation of cerebrovascular status impossible.

Shalit and Umansky (1977) reported observations of selected activities in 21 comatose patients with brain edema secondary to head injury or brain gliomas. ICP was measured by both intraventricular cannula and subdural transducer. Increases and decreases in ICP after suctioning patients were found to correlate with changes in end tidal CO2 levels. When CO2 levels decreased, then ICP decreased and vice versa. Mean arterial blood pressure and cerebral perfusion pressure alterations were not reported.

Effects of Suctioning on Cerebrovascular Status

Parsons and Shogan (1984) studied the effects of an endotracheal tube suctioning/manual hyperventilation (ETTS/MH) procedure upon the cerebrovascular status of 20 severe closed head injured adults and children. All patients had stable cardiovascular and respiratory status and had subarachnoid bolts in place for ICP measurement. Baseline ICP's were 0 to 20 mm Hg. Dependent variables in this pilot study were ICP, CPP, MABP, and heart rate. They found that CPP levels were maintained at levels greater than 70mm Hg, which is adequate to maintain an adequate blood flow to the brain, in spite of significant increases in mean ICP during the ETTS/MH procedure. Autoregulation was

determined to be intact by parallel changes between mean arterial blood pressure and ICP. The findings suggest that the ETTS/MH procedure could be safely performed upon patients with severe closed head injuries with baseline ICP measurements of 0 to 20mm Hg.

A follow-up study by Parsons, Vollmer, Crosby, and Holley-Wilcox (1983) was done evaluating the effects of a modified ETTS/MH procedure in 15 closed head injured adult patients. All subjects were required to be without severe pulmonary-thoracic complications. The subjects were split into two groups of seven, one with resting mean ICP < 9mm Hg and the other with resting mean ICP >9 mm Hg. Following the second and third suction passes MH was increased from 30 to 60 seconds. Progressive decreases in mean ICP were associated with the 60 second MH periods. These results suggest that the increased MH time is more effective in controlling suction induced mean ICP increases in closed head injured patients with baseline mean ICP less than 20 mm Hg.

Fisher, Frewen, and Swedlow (1982) studied nine children (9 months to 12 years) admitted to a pediatric ICU with increased ICP measured by a subarachnoid screw. The purpose of the study was to determine the separate contribution to changes in ICP of tracheal stimulation and changes in PaCO2. Each patient was studied twice, once with and once without suctioning. Two minutes prior to the start

of the protocol, the FiO2 was increased to 1.0. Sixty seconds before the start of suctioning the patient was hyperventilated by increasing the rate by 25%. The patient was then disconnected from the ventilator for 30 seconds. In one trial the patient was suctioned for 30 seconds and in the other the patient was apneic. Mean ICP increased 5 mm Hg and MABP increased 10mm Hg with no significant changes in CPP during the suctioning trial. There was no significant change in these parameters during the nonsuctioning trials. End-tidal CO2 (ETCO2), measured with an infrared carbon dioxide analyzer, increased during both trials with the conclusion that increased ICP was due to tracheal stimulation rather than increased PaCO2.

Summary

Following a review of literature, it is evident that research in the area of the effect of nursing activities on cerebrovascular status has provided baseline information that can be applied to clinical practice. The majority of reported research has been descriptive of the influence of various care activities on ICP.

Although suctioning is frequently cited as a potent stimulus to further increases in ICP in patients with intracranial hypertension, little systematic study has documented the actual changes in ICP and CPP with controlled suctioning protocols and the impact of cardiovascular parameters on these measures. Little is known about the

mechanisms by which activities such as suctioning effect demands upon the craniospinal system, or what the best predictors are of individuals who will respond adversely to such demands.

When performing physiological studies, it is important to recognize that cranial pathologies vary and that the patient's primary diagnosis may have an affect on response to endotracheal suctioning (Rudy, 1986). Small and varied sample sizes have limited the generalizability of results to the head injured population in general. This study is designed to build on the work of Parsons and colleagues in an attempt to contribute to the developing body of knowledge related to the effects of endotracheal suctioning and manual hyperventilation on cerebrovascular status of head injured patients.

CHAPTER III

METHODOLOGY

Statement of Purpose

This quasi-experimental study was designed to determine the effects of endotracheal tube suctioning (ETTS) with manual hyperventilation (MH) on the cerebrovascular status of severe closed head injured patients. Parameters monitored to examine the changes in cerebrovascular status during the suctioning procedure were cerebral perfusion pressure (CPP), intracranial pressure (ICP), mean arterial blood pressure (MABP), and heart rate (HR).

This chapter describes the research design, sample, operational definitions and data analysis used to implement this study. Methodological assumptions and limitations of the study are also addressed.

Research Design and Procedure

The quasi-experimental study utilized a within-subject repeated measure design. A convenience sample of 20 patients with closed head trauma was studied. All subjects met the following criteria for inclusion in the study:

1. Admission to Maryland Institute for Emergency Medical Services Systems (MIEMSS), Neurotrauma Unit (NTU) or Critical Care Recovery Unit (CCRU).
2. Subarachnoid pressure bolt or intraventricular catheter in place for monitoring intracranial pressure (ICP).

3. Arterial catheter for monitoring of blood pressure.
4. Electronically monitored heart rate.
5. Intubated and ventilated by a mechanical ventilator.
6. Mean arterial blood presssure greater than 50mm Hg.
7. Glasgow Coma Scale (GCS) rating of greater than or equal to 3 and less than or equal to 10.
8. PaO2 of 70mm Hg or greater.
9. PaCO2 of 28mm Hg to 32mm Hg.
10. FiO2 no greater than 0.4.
11. Positive End Expiratory Pressure (PEEP) less than 15cm H2O.
12. Body temperature between 35 and 38 degrees Centigrade.

The standardized ETTS/MH protocol was the independent variable. The dependent variables were HR, MABP, CPP, and ICP. The dependent variables for each subject was continuously monitored during the protocol using a four-channel data recorder. ICP was monitored via subarachnoid bolt or fiberoptic (Camino) catheter.

All data was collected by a two person nursing research team utilizing a standardized protocol. The frequency of measurement and treatment protocols were designed to reflect usual clinical situations. Data was collected during regularly scheduled suctioning times.

Before the protocol was initiated, the nurse assigned to the subject was consulted to determine the time of the last suctioning procedure. A convenient time was selected by the research team and staff nurse to perform the following protocol.

1. Apply pulse oximetry sensor to fingertip.
2. Attach and calibrate Data Collection Recorder to bedside monitoring and pulse oximetry.
3. Begin recording of baseline data on recorder.
4. Record baseline data for 5 minutes.
   a. Channel One: Heart rate
   b. Channel Two: Oxygen saturation
   c. Channel Three: Blood pressure
   d. Channel Four: Intracranial pressure
5. Mark recorder at end of 5 minute baseline.
6. Manually hyperventilate (MH) for 30 seconds at 1.0 FiO2 using a Puritan-Bennett PMR-2 manual resuscitation bag with 640 ml oxygen reservoir and supplemental O2 at a flow rate of 15 liters/minute.
7. Endotracheal tube suctioning (ETTS) for 10 seconds using a standard 14 FR straight suction catheter and negative pressure of 120mm Hg..
8. MH for 45 seconds.
9. ETTS for 10 seconds.
10. MH for 45 seconds.
11. ETTS for 10 seconds.

12. MH for 30 seconds.
13. Reconnect to ventilator and mark recorder.
14. Record post-procedure data for 5 minutes.
15. End protocol.

All data was recorded on a graphic tracing utilizing a Mennen four-channel data recorder. All channels were calibrated using a standardized procedure (Appendix A). The five minute pre and post-procedure measurements were divided into one-minute intervals and the ETTS/MH portion of the protocol was divided into 15 second intervals for purposes of evaluation. A stop watch was used to time the various intervals during the protocol. A total of 23 data points were calculated for each dependant variable with a total protocol time of 13 minutes.

Patients were not placed in a predetermined position prior to the start of the protocol. The protocol was performed with the head of the bed up at least 30 degrees and the patient's position was not changed once the protocol was initiated. If a rise in ICP occurred requiring a position change, the data collection was stopped. During baseline recordings, no direct-contact patient care activities were performed and the patient was not hyperoxygenated during this time. If at any time during the procedure the ICP exceeded 25mm Hg for a sustained period of time, the procedure would have been immediately discontinued.

This study was conducted as part of a larger investigation entitled "The Effects of Selected Activities on Intracranial Pressure in Closed Head Injured Patients". Both members of the research team collected data for a similar protocol prior to collecting data for this study. Based on the familiarity with the instrumentation and clinical environment, a pilot study was not deemed necessary.

The professional nursing staff of the NTU and CCRU where the study was conducted were given inservices prior to its implementation. They were informed of the purpose of the study, research activities at the bedside, and their involvement in the data collection process.

Sample

A convenience sample of 20 subjects from MIEMSS was studied. Twelve of the subjects were observed in the NTU and eight were studied in the CCRU. Both of these areas are within MIEMMS and are critical care units with identical patient monitoring systems.

Patients included in this study had a documented diagnosis of closed head injury (See Table 1). A closed head injury was defined as a mechanical blow to the head with cerebral trauma demonstrated by decreased level of consciousness without penetration of the dura mater. The severity of head injury was quantified using the Glasgow Coma Scale (GCS) (Appendix B) developed by Teasdale and

Jennett (1974). Three functions are assessed (eye opening, best verbal response, and best motor response) and a total score is calculated by adding the highest patient response in each area. GCS scores can range from 15, considered normal, to three, indicating a severely depressed level of consciousness. Subjects had GCS scores ranging from 3 to 14 on admission and from 3T to 10T at the time of data collection. T indicates the patient was intubated at the time of assessment and therefore best vebal response could not be tested.

All the subjects had an intracranial monitoring device in place. Subarachnoid bolts were used in 18 of the cases, and a fiberoptic catheter was used in two patients. All subjects were on mechanical ventilators and had arterial lines for arterial blood pressure monitoring.

Table 1

Patient's Etiology of Injury, Head Injury Diagnosis, Glasgow Coma Scale Scores and Accompanying Injuries

Patient Code Number	Etiology of injury	Head Injury Diagnosis	Admission GCS	GCS at time Observed	Accompanying Injuries
01	Pedestrian struck by vehicle	CHI Right frontal edema Left temporal-parietal SDH Contusions and swelling with shift.	4	4T	Left femur fracture Left scapula fracture Lacerations Ruptured spleen/spleenectomy Right pneumothorax/chest tube
02	Fall from bike	CHI, Basilar skull fracture, Right temporal lobe Subarachnoid hemorrhage, inoperable subdural hematoma, comminuted left occipital-temporal skull fracture.	7	7T	Rib cage derangement
03	Motorcycle accident	CHI, Left epidural hematoma.	14	4T	Pelvic fracture Anal tear and rectal perforation S/P right trannsverse colostomy Lacerations
04	Motor vehicle accident	CHI, intracerebral hematoma, frontal hemorrhage, petrous fracture.	7	9T	Full thickness laceration forehead
05	Fall down stairs	CHI, subdural hematoma, bilateral contusions.	14	10T	None
06	Motorcycle accident	CHI, intracerebral hemorrhage, contusion internal capsule, contusion brainstem.	4	4T	Mandibular fracture Right zygomatic fracture Frontal sinus fracture Scalp laceration Abrasions of face, shoulder, chest
07	Motorcycle accident	CHI, temporal-parietal contusion, subarachnoid bleed.	14	5T	Left femur fracture Left tibia fracture Splenic lacerations (2) S/P spleenorophy Right peri-orbital ecchymosis
09	Motor vehicle accident	CHI, temporal contusion.	11	6T	Fracture maxilla (Lefort III) Fracture mandible-S/P ORIF Multiple lacerations

(Table continued)

09	Motor vehicle accident	CHI, frontal contusion.	10	10T	C5 wedge fracture
10	Pedestrian struck by vehicle	CHI, frontal-temporal contusion.	13	10T	Pelvic ring disruption Left tibia fracture Right humerus fracture Multiple facial/scalp abrasions
11	Motor vehicle accident	CHI, temporal contusion, thalamic contusion.	6	5T	Lacerated spleen-S/P spleenectomy Cheek laceration
12	Motorcycle accident	CHI	11	10T	Open fracture right humerus Left tibia fracture Multiple facial fractures Multiple chest wall and neck lacerations
13	Stabbing	Stab wound to brain stem.	5	3T	Right chest stab wound with hemopneumothorax Right abdominal wound with torn pancreaticoduodenal artery Right hemicolectomy and colostomy
14	Motor vehicle accident	CHI, temporal-parietal contusion.	6	7T	Superior and inferior pubic rami fracture Splenic laceration-S/P spleenectomy Liver laceration
15	Motor vehicle accident	CHI, frontal-temporal contusion.	11	8T	Comminuted/open fracture left femur Fracture left tibial plateau Fracture metacarpals Open/comminuted spiral fracture right tibia/fibula
16	Motor vehicle accident	CHI, negative CT scan.	9	3T	Comminuted fracture T12 C7 fracture Transverse process fracture L1-2 Left scapula fracture Left hemopneumothorax and flail chest Multiple rib fractures
17	Motor vehicle accident	CHI	3	4T	Right femur fracture Racoon eyes-Right Right zygoma fracture Spleen laceration-S/P splenorraphy
18	Motor vehicle accident	CHI, frontal-temporal contusion, diffuse edema.	7	8T	Open fracture/dislocation right ankle Hemoperitoneum secondary to splenic lac. S/P spleenorraphy Right occipital scalp flap laceration

(Table continued)

19	Pedestrian struck by vehicle	CHI, frontal-temporal contusion, subarachnoid hemorrhage with diffuse swelling.	3	5T	Left knee fracture Hematuria (IVP & cycto neg) Scalp laceration Diastasis of symphysis pubis	36
20	Assault	CHI, intracranial hemmorhage with mass effect.	6	10T	S/P craniotomy (POD #1)	

Operational Definitions and Instrumentation

Intracranial Pressure

Intracranial Pressure (ICP) is the force exerted by the components within the skull (brain tissue, blood, and cerebrospinal fluid). ICP was measured by use of a subarachnoid bolt (Richmond screw). Two patients had fiberoptic sensors in place. These measuring devices were placed by the physician and were deemed medically necessary to monitor the patient's critical condition. The screw is inserted through a drill hole in the skull. The dura is pierced and the bolt is projected onto the arachnoid surface of the brain. It senses and transmits pressure changes in the CSF (Chapman, 1983). A transducer is secured to the measuring device and pressure signals are transmitted to a MENNEN monitor (480 series, number 426) which displays a continuous digital and waveform readout of the ICP. The ICP measuring system was calibrated using sterile technique, immediately prior to the start of data collection during the data recorder calibration and hook-up procedure.

Normal ICP ranges from 0 to 15 mm Hg. Sustained pressures above 20 mm Hg are considered abnormally elevated (Miller, Becker, Ward, Sullivan, Adams, & Rosner, 1977).

Mean Arterial Blood Pressure

Mean arterial blood pressure was monitored via an intra-arterial catheter which was inserted by a physician as part of the routine critical care patient management. A

transducer is attached to the catheter and pressure signals are transmitted to the same MENNEN monitor as above which displays a continuous digital and waveform readout of the blood pressure. The monitor system is capable of providing systolic/diastolic or mean values in digital form. The measuring system was calibrated immediately prior to the start of data collection using sterile technique.

Heart Rate

Heart rate was determined by electrocardiogram. Standardized three-lead electrode placement using pre-gelled electrodes was utilized. A three lead cable was attached to the MENNEN monitoring system. Heart rate was recorded in Lead II on the MENNEN four-channel recorder.

Oxygen Saturation

Oxygen saturation was monitored continuously via a Nellcor N-200 pulse oximetry unit. A digital NELLCOR sensor was placed on the subject's fingertip. The Nellcor N-200 is calibrated to read oxyhemoglobin saturation (% SaO2) of functional hemoglobin. Accuracy was established at +/- 2 digits for saturation readings of 70 to 100 percent (Nellcor, Inc., 1987).

Puritan-Bennett PMR-2 Manual Resuscitation Bag

Manual hyperventilation was delivered using the Puritan-Bennett PMR-2 manual resuscitation bag. With reservoirs attached, the PMR-2 achieves a mean fractional delivered oxygen concentration (FDO2) >0.90 at a flowrate of

15 liters/minute (Barnes & Watson, 1982). The manual resuscitation bag was supplied with O2 from a calibrated flow meter attached to a source of O2 at 50 psi pressure.

Mennen Series 480 Patient Monitoring System

Heart rate, arterial blood pressure, and intracranial pressure were measured using the Mennen Series 480 bedside patient monitoring system with established validity and reliability. The Biomedical Engineering department calibrated all Mennen modules prior to the initiation of data collection for the study. Heart rate accuracy was determined to be +/- 3 beats per minute or +/- 2% of the reading, whichever is greater. Pressure module output drift was documented at less than +/- one percent full scale per 24 hours. Accuracy was reported to be +/- 3 mm Hg or two percent of the reading (whichever is greater) at the range setting of 300 mm Hg (used for blood pressure monitoring), to +/- 1 mm Hg in the 25 mm Hg range (used for ICP monitoring) (Mennen Medical, Inc., 1979).

Data Collection Tool

A data collection tool was developed for use in this study (Appendix C). The instrument was composed of two parts. Part one contained the patient history including Subject Identification Number, and date and time of data collection. Demographic data collected included age, sex, race, and date of injury. Admission data included Blood alcohol levels, admission GCS score, and primary diagnosis.

Extent of injuries was identified by system (Head injury, spinal cord injury, skeletal, cardiac, vascular, respiratory, renal, gastrointestinal, and reproductive).

Part two was used to document physiological parameters at the time of data collection. These included days since injury, current GCS, ICP for last eight hours (range and mean was calculated from this data), Date ICP monitoring initiated and the type of monitoring device, most recent blood gas results, ventilator settings, patient temperature, and the current drug regimen (drug, dose, and time of last dose).

Protocol data was continuously recorded utilizing the Mennen four channel data recorder. The recorder strips were evaluated by the primary investigator following the completion of the protocol and data was retrieved from these strips for analysis.

Human Rights Protection

The anonymity of the subjects entered into this study was maintained by use of an identification code number for each patient. No patient names or other identification methods were used.

The suctioning protocol utilized for this study was the standard suctioning practice utilized for patient care in this institution. Absence of the introduction of any experimental treatment to any of the subjects negated the need for a consent form to be used. This protocol was

approved by the Human Rights Committee of the University of Maryland, the Human Rights Committee of the Maryland Institute for Emergency Medical Services Systems (MIEMSS), and the MIEMSS Nursing Research Committee.

Data Analysis

Data collection produced interval level data for evaluation of the effects of endotracheal suctioning and manual hyperventilation on closed head injured patients. The data analysis was done in several steps.

During data collection, the data recorder strips were marked to identify protocol intervals (pre-procedure baseline, manual hyperventilation, suctioning, and post procedure observation). The data recorder strips were standardized so that every strip had the same dependent variable recorded on each channel (Appendix A). The recorder documented all four channels simultaneously. Channel one recorded heart rate, channel two recorded oxygen saturation, channel three recorded blood pressure, and channel four recorded intracranial pressure. The recorder speed was set at 5mm/second.

Following completion of the protocol, the data recorder strips were read. The waveform reproduced on the paper was exactly as that observed on the bedside monitoring equipment. The primary investigator read every strip to maintain reliability during this process. Data recorder strips were analyzed following a standardized format.

1. The paper is divided into large and small blocks, identical to EKG paper Appendix D). There are four strips per page, one above the other. Each large block contains five small blocks and the paper is ten large (50 small) blocks tall.

2. The range for pressure channels which was set during calibration determined the value of each block for measurement purposes as follows:

   a. Heart Rate - With paper speed at 5mm/second, each area between the slashes at the top of the paper represents 15 seconds instead of the traditional three second strip.

   b. Oxygen Saturation - Each large block on the vertical axis represents 10% O2 saturation and each small block represents 2% O2 saturation. The bottom of the strip represents absolute zero.

   c. Blood Pressure - Each large block on the vertical axis represents 30mm Hg and each small block represents 6mm Hg. The bottom of the strip represents absolute zero.

   d. Intracranial Pressure - Each large block represents 5mm Hg and each small block represents 1mm Hg. The bottom of the strip represents absolute zero.

Using the above criteria, each strip was evaluated to determine a value for each of the dependent variables across

all 23 intervals (See Figure 2). The strip was manually highlighted to identify the protocol intervals as follows:

Interval 1 - Baseline minute 1 (1 minute)

Interval 2 - Baseline minute 2 (1 minute)

Interval 3 - Baseline minute 3 (1 minute)

Interval 4 - Baseline minute 4 (1 minute)

Interval 5 - Baseline minute 5 (1 minute)

Interval 6 - MH 1a (15 seconds)

Interval 7 - MH 1b (15 seconds)

Interval 8 - ETTS 1 (15 seconds)

Interval 9 - MH 2a (15 seconds)

Interval 10 - MH 2b (15 seconds)

Interval 11 - MH 2c (15 seconds)

Interval 12 - ETTS 2 (15 seconds)

Interval 13 - MH 3a (15 Seconds)

Interval 14 - MH 3b (15 seconds)

Interval 15 - MH 3c (15 seconds)

Interval 16 - ETTS 3 (15 seconds)

Interval 17 - MH 4a (15 seconds)

Interval 18 - MH 4b (15 seconds)

Interval 19 - Post-procedure minute 1 (1 minute)

Interval 20 - Post-procedure minute 2 (1 minute)

Interval 21 - Post-procedure minute 3 (1 minute)

Interval 22 - Post-procedure minute 4 (1 minute)

Interval 23 - Post-procedure minute 5 (1 minute)

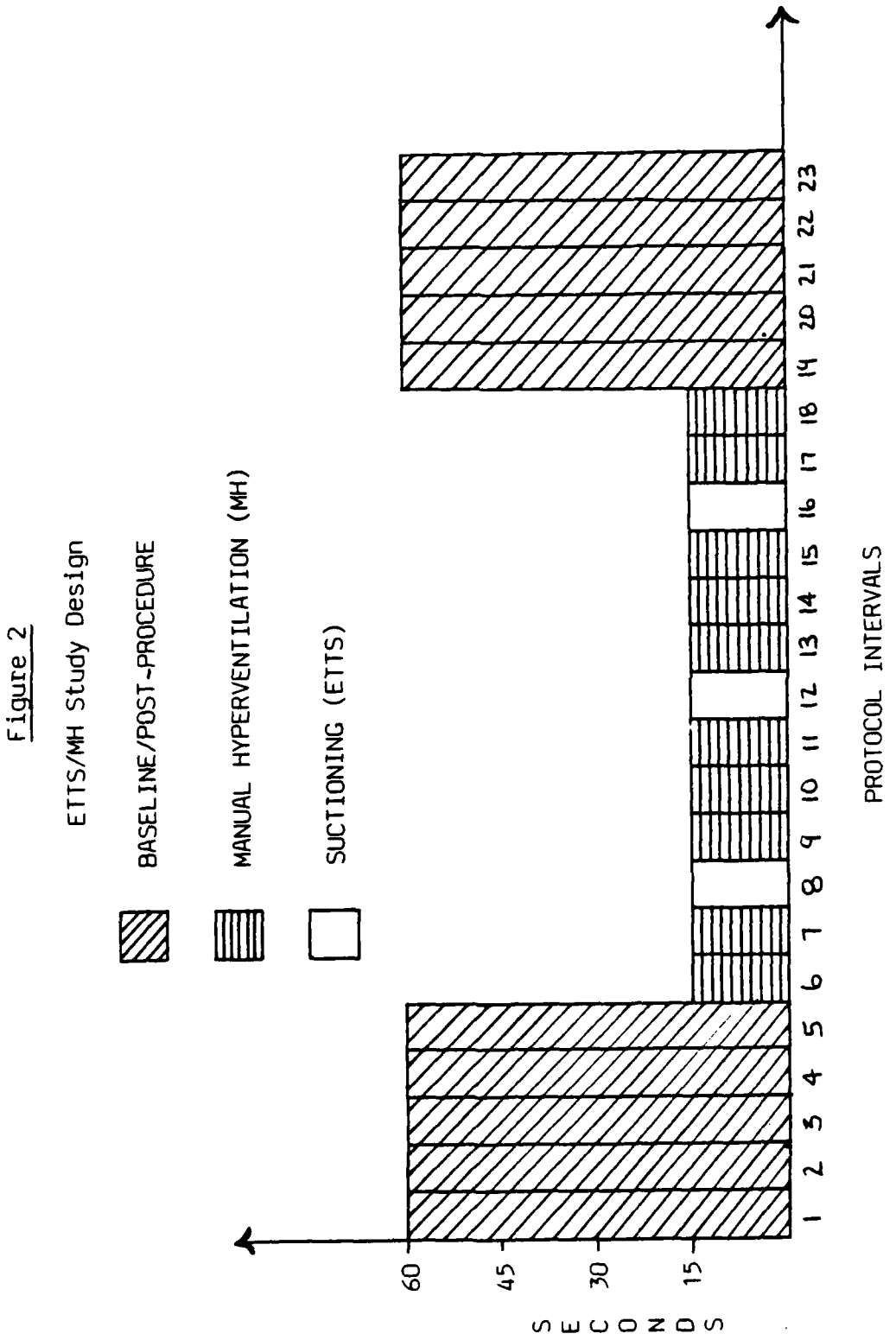
The actual ETTS/MH portion of the strip was divided into 15

second intervals so that variability within these time frames could be analyzed if necessary. Suctioning was limited to ten seconds but the time frame of 15 seconds was utilized for standardization of time frames and to allow for disconnecting and reconnecting the patient to the manual ventilation bag.

The values for each interval were determined using the criteria above. Because very little variability was noted, heart rate was calculated by counting the number of beats in a 15 second time period and multiplying by four. Oxygen saturation levels revealed very little variability and the value was determined by taking the value present at the end of each interval.

Blood pressure and intracranial pressure waveforms revealed great variability within certain time intervals. Therefore, the value for each interval was determined by taking the highest and lowest value within that interval and averaging these values. The highest and lowest diastolic and systolic blood pressure values were each averaged separately to arrive at a value for that particular time interval. The same was done for intracranial pressure. An arithmetic mean was not done on these variables because not all waveforms were articulated clearly enough to perform this function.

Figure 2


ETTS/MH Study Design
BASELINE/POST-PROCEDURE
MANUAL HYPERVENTILATION (MH)
SUCTIONING (ETTS)
60
45
30
15
SECONDS
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
PROTOCOL INTERVALS

The value for each of the 23 time intervals in all four dependent variables was then entered into a LOTUS 1-2-3 spreadsheet for the purpose of calculating mean arterial blood pressure and cerebral perfusion pressures. The mean arterial blood pressure was calculated by the computer using the following equation: MABP = 1/3 (Systolic BP - Diastolic BP) + Diastolic BP (Cardona et al, 1988, p. 373). MABP and ICP values were utilized to calculate CPP. CBF adequacy was determined by evaluating the CPP. A CPP of 50 to 130 mm Hg is indicative of adequate CBF (Miller, 1982). The cerebral perfusion pressure was calculated by the computer using the following equation: CPP = MABP - ICP (Cardona et al, 1988, p. 373).

The Statistical Package for the Social Sciences (1983) was used for data analysis. The SPSS-X MANOVA was the statistical package utilized for data analysis. Level of significance for these analyses was set at .05. Each dependent variable was analyzed separately.

First, repeated measures analysis of variance (ANOVA) was calculated across all 23 time intervals for the group average for each dependent variable. If significant variability appeared across all intervals, additional ANOVA calculations were conducted on related time intervals (baseline and post-procedure, all suctioning intervals, all hyperventilation intervals) to determine if significant variability existed within these time frames. If

significant variability was not found, a mean was computed for that time interval.

Demographic data was summarized with descriptive statistics for assessment of relationship to the results of the study. Demographic data is presented in Chapter IV.

Limitations of the Study

1. Generalizability of the results is limited to the head injured population in this study.

2. Medication types and dosages varied between subjects and their affect on ICP or MABP could be construed as a confounding variable.

3. Patient agitation during the suctioning procedure based on varying levels of sedation and the resultant effect on ICP and MABP constituted a confounding factor.

4. The critical care environment may cause a change in a dependent variable unrelated to the treatment, although this was controlled as much as possible.

It is believed that even with these limitations, the study will produce results that are pertinent to the developing body of knowledge related to nursing care of closed head injured individuals.

The results of this study will be presented to medical and nursing personnel at MIEMSS. The results will be submitted for publication in a nursing journal.

CHAPTER IV

PRESENTATION OF RESULTS

Statement of Purpose

The purpose of this study is to increase the body of knowledge on the effect of nursing activities on head injured patients. This study examines the effects of endotracheal suctioning with manual hyperventilation (ETTS/MH) on the cerebrovascular status of severe closed head injured patients.

Statistical analysis of the data collected and demographic and clinical characteristics of the sample will be reported. Each dependent variable will be discussed separately.

Description of Sample

The mean age of the study sample was 27.45 years. Seventy percent of the sample were males (14) and 30% were female (6). Sixteen (80%) of the subjects had been involved in a motor vehicle accident, two (10%) were hospitalized for falls, and two (10%) as a result of assaults. Eighty percent of the sample was caucasian (16) and 20% were black (4) (See Table 2). Forty percent of the group tested positive for drugs and/or alcohol in their serum upon admission.

TABLE 2

Summary of Demographic Data

n=20

Age		
	Mean:	27.45
	S.D.:	14.06
	Range:	16-73
Sex		
	Male:	14
	Female:	6
Race		
	Caucasian:	16
	Black:	4
Monitoring Device		
	Subarachnoid Bolt:	18
	Fiberoptic Catheter:	2

Admitting GCS scores ranged from 3 to 14 with 55% of the sample scoring less than eight on the scale. The GCS scores assessed at the time of data collection are all accompanied with a T indicating that all the subjects were intubated and mechanically ventilated at the time of assessment. Seventy percent of the GCS scores calculated at the time of data collection were less than 8T (See Table 3).

TABLE 3

GCS Scores Upon Admission and During Data Collection

n=20

Admission	
Mean:	8.30
S.D.:	3.84
Range:	3-14
During Data Collection	
Mean:	6.60T
S.D.:	2.60
Range:	3T-10T

Baseline ICP values ranged from 0.6-20.6 mm Hg with 80% of the sample falling within the normal range of 0-15mm Hg (Table 4). This indicates that the subjects were low on the intracranial pressure-volume curve which would suggest increased tolerance of transient elevations in ICP during the protocol (Rudy, 1986; Smith, 1983).

TABLE 4

Baseline Intracranial Pressures

n=20

Mean:	10.40
S.D.:	5.37
Range:	0.6-20.6

Although most of the patients had sedation ordered, only eight (40%) had received such medication within 4 hours prior to data collection. Five had received Sublimaze and three had received Sodium Pentothal.

Analysis of the Research Question

The results of statistical analysis will be presented individually for each dependent variable in the following order: Heart rate (HR), mean arterial blood pressure (MABP), intracranial pressure (ICP), cerebral perfusion pressure (CPP), and oxygen saturation (O2 SAT).

Repeated measures analysis of variance (ANOVA) was calculated across all 23 time intervals using the group mean for each dependent variable. An ANOVA Summary Table is provided for each dependent variable where statistically significant differences were identified. The means and standard deviations are reported in Appendices E-H and graphically in Appendix I. In the presence of significant variability across all intervals, additional ANOVA calculations were conducted on related time intervals (baseline and post-procedure, all suctioning intervals, and all hyperventilation intervals) to determine if significant variability existed within these time frames. A mean was computed for each time interval which did not have statistically significant variation which will be used to address the study research question. Interval means are signified with (M) in the Tables.

Heart Rate

An analysis of variance for repeated measures was done to determine significant changes in heart rate during the ETTS/MH protocol. To determine this, an ANOVA was done across all 23 time intervals with a significant F value of 4.19 (p=.000) (Table 5). The means and standard deviations are presented graphically in Figure 3.

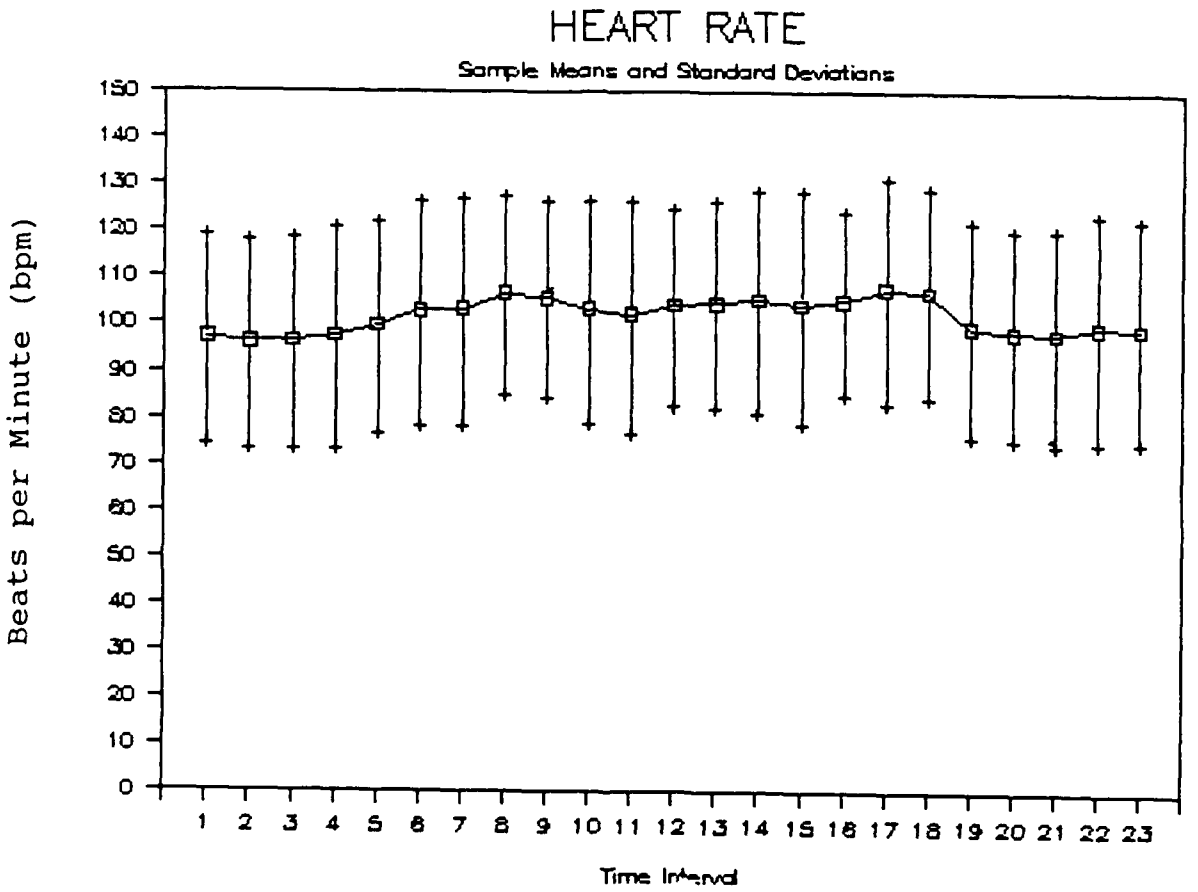
Table 5

Repeated Measures Analysis of Variance
Summary Table for HR across 23 time intervals

Source	df	SS	MS	F	Sig. of F
Within Cells	418	26548.43	63.51		
TIME	22	5858.17	266.28	4.19	.000

Figure 3

Graphic Representation of
Heart Rate Means and Standard Deviations
Across All 23 Time Intervals



Baseline: Intervals 1-5

ETTS: Intervals 8, 12, & 16

MH: Intervals 6-7, 9-11, 13-15, & 17-18

Post Procedure: Intervals 19-23

ANOVA's were done to compute variable means for similar time periods (baseline, suctioning, MH, and recovery phase) (Table 6).

Table 6

Repeated Measures ANOVA
Heart Rate Time Intervals

Intervals	F	Sig. of F
1-5, 19-23	1.46	.166
1-5 (M), 6-18, 19-23(M)	2.67	.001*
MH 6-7, 9-11, 13-15, & 17-18	1.76	.078
Suctioning 8, 12, & 16	.37	.695
1-5 (M), Suctioning (M), 19-23 (M)	3.55	.010*
MH 6-7, Suction 8	1.14	.330
MH 9-11, Suction 12	1.24	.302
MH 13-15, Suction 16	.10	.958
Suction 16, MH 17-18	.74	.484

*significant ($p < .05$) (M)=mean of interval values

Because there was not a significant difference within time frames of baseline, MH, suctioning, and recovery period heart rates, means were computed for each of these time frames and an ANOVA was done to examine the overall effect of the procedure on heart rate (Table 7).

Table 7

Repeated Measures ANOVA
Summary Table for Heart Rate

Source	df	SS	MS	F	Sig. of F
Within cells	57	3169.18	55.60		
TIME	3	936.51	312.17	5.61	.002

The means and standard deviations are presented graphically in Figure 4. A Tukey HSD Test was performed on the mean values to determine which intervals were significantly different. The time intervals in which these significant differences occurred were from baseline to suctioning (ETTS), baseline to manual hyperventilation (MH), ETTS to recovery period, and MH to recovery period (Table 8).

Table 8

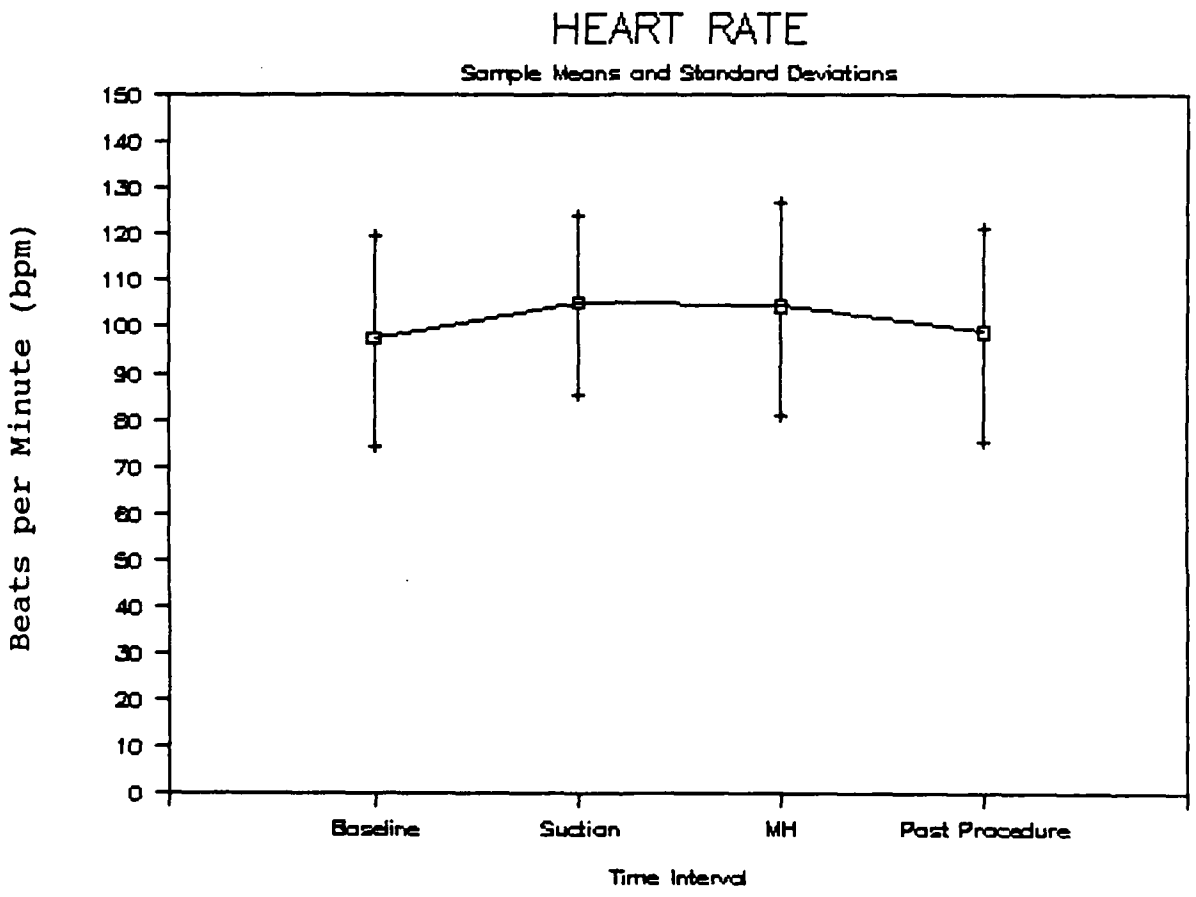
Significant Time Interval Differences
Heart Rate

	Base	Suction	MH	Recovery
Mean	97.40	105.23	104.57	98.92
Base		*	*	
Suction	*			*
MH	*			*

*significant ($p < .05$)

Figure 4

Graphic Representation of
Heart Rate Means and Standard Deviations
For Major Time Intervals



Mean Arterial Blood Pressure

An analysis of variance for repeated measures was done to determine significant changes in mean arterial blood pressure (MABP) during the ETTS/MH protocol. To determine this, an ANOVA was done across all 23 time intervals with a significant F value of 5.25 (p=.000) (Table 9).

Table 9

Repeated measures Analysis of Variance
Summary Table for MABP across 23 time intervals

Source	df	SS	MS	F	Sig. of F
Within Cells	418	17210.91	41.17		
TIME	22	4759.96	216.36	5.25	.000

In order to compute means for this variable, ANOVA's were done on various similar time periods (baseline, suctioning, MH, and recovery phase) (Table 10). The means and standard deviations are presented graphically in Figure 5.

Table 10

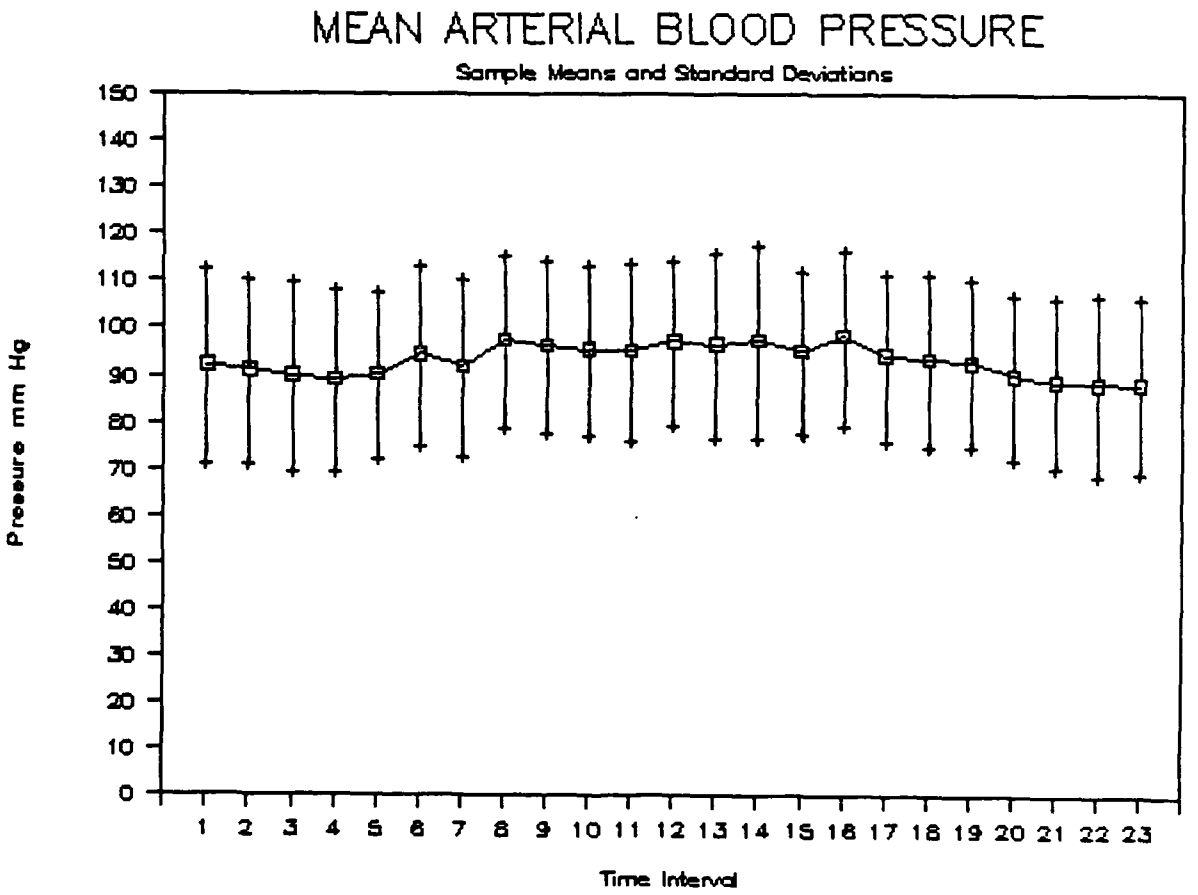
Repeated Measures ANOVA
Mean Arterial Blood Pressure Time Intervals

Intervals	F	Sig. of F
1-5, 19-23	1.55	.135
1-5 (M), 6-18, 19-23 (M)	3.09	.000*
MH 6-7, 9-11, 13-15, & 17-18	1.68	.096
Suctioning 8, 12, & 16	.21	.813
1-5 (M), Suctioning (M), 19-23 (M)	5.53	.001*
MH 6-7, Suction 8	2.87	.069
MH 9-11, Suction 12	.95	.421
MH 13-15, Suction 16	1.39	.254
Suction 16, MH 17-18	4.75	.014*

*significant ($p < .05$) (M)=mean of interval values

Figure 5

Graphic Representation of
MABP Means and Standard Deviations
Across All 23 Time Intervals



Baseline: Intervals 1-5

ETTS: Intervals 8, 12, & 16

MH: Intervals 6-7, 9-11, 13-15, & 17-18

Post Procedure: Intervals 19-23

Because there was not a significant difference within time frames of baseline, MH, suctioning, and recovery period mean arterial blood pressure values, means were computed for each of these time frames and an ANOVA was done to examine the overall effect of the procedure on mean arterial blood pressure (Table 11).

Table 11

Repeated Measures ANOVA
Summary Table for Mean Arterial Blood Pressure

Source	df	SS	MS	F	Sig. of F
Within Cells	57	1826.21	32.04		
TIME	3	870.5	290.17	9.06	.000

The means and standard deviations are presented graphically in Figure 6. A Tukey HSD Test was performed on mean values to determine which intervals were significantly different. The intervals in which these significant differences occurred were from baseline to ETTS, baseline to MH, ETTS to recovery period, and MH to recovery period (Table 12).

Figure 6

Graphic Representation of
MABP Means and Standard Deviations
for Major Time Intervals

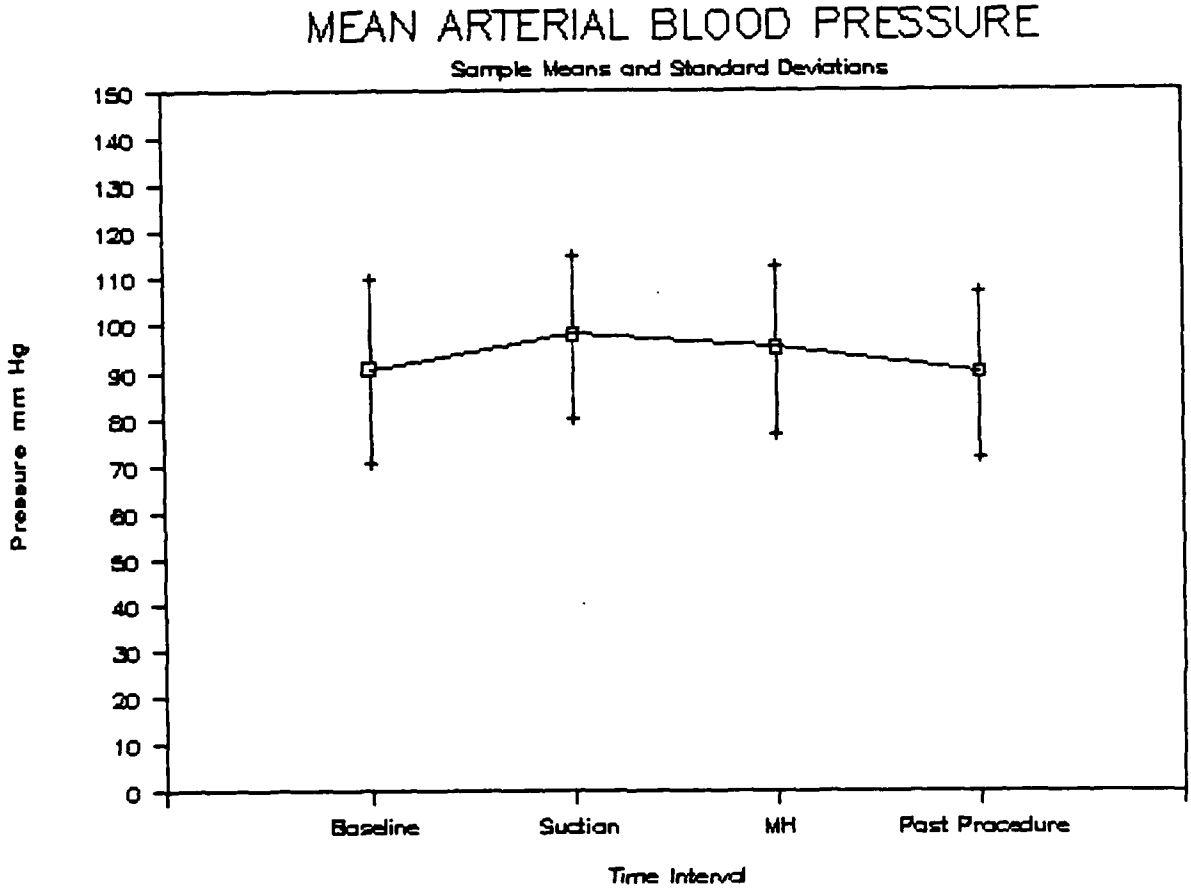


Table 12

Significant Time Interval Differences
Mean Arterial Blood Pressure

	Base	Suction	MH	Recovery
Mean	90.60	97.80	95.12	89.74
Base		*	*	
Suction	*			*
MH	*			*

*significant ($p < .05$)

Intracranial Pressure

An analysis of variance for repeated measures was done to determine significant changes in intracranial pressure (ICP) during the ETTS/MH protocol. To determine this, an ANOVA was done across all 23 time intervals with a significant F of 3.56 (p=.000) (Table 13). The means and standard deviations are presented graphically in Figure 7.

Table 13

Repeated Measures Analysis of Variance
Summary Table for ICP across 23 time intervals

Source	df	SS	MS	F	Sig. of F
Within Cells	418	9497.48	22.72		
TIME	22	1778.34	80.83	3.56	.000

Thirteen (65%) subjects experienced transient ICP spikes of greater than 20 mm Hg during the suctioning intervals of the protocol. The spike pattern lasted from 3 to 6 seconds in duration. There was no relationship between sedation and presence of spiking during suctioning. Two patients experienced only one spike during the entire protocol, four subjects experienced two spikes during the protocol, and seven experienced spikes during every suctioning pass.

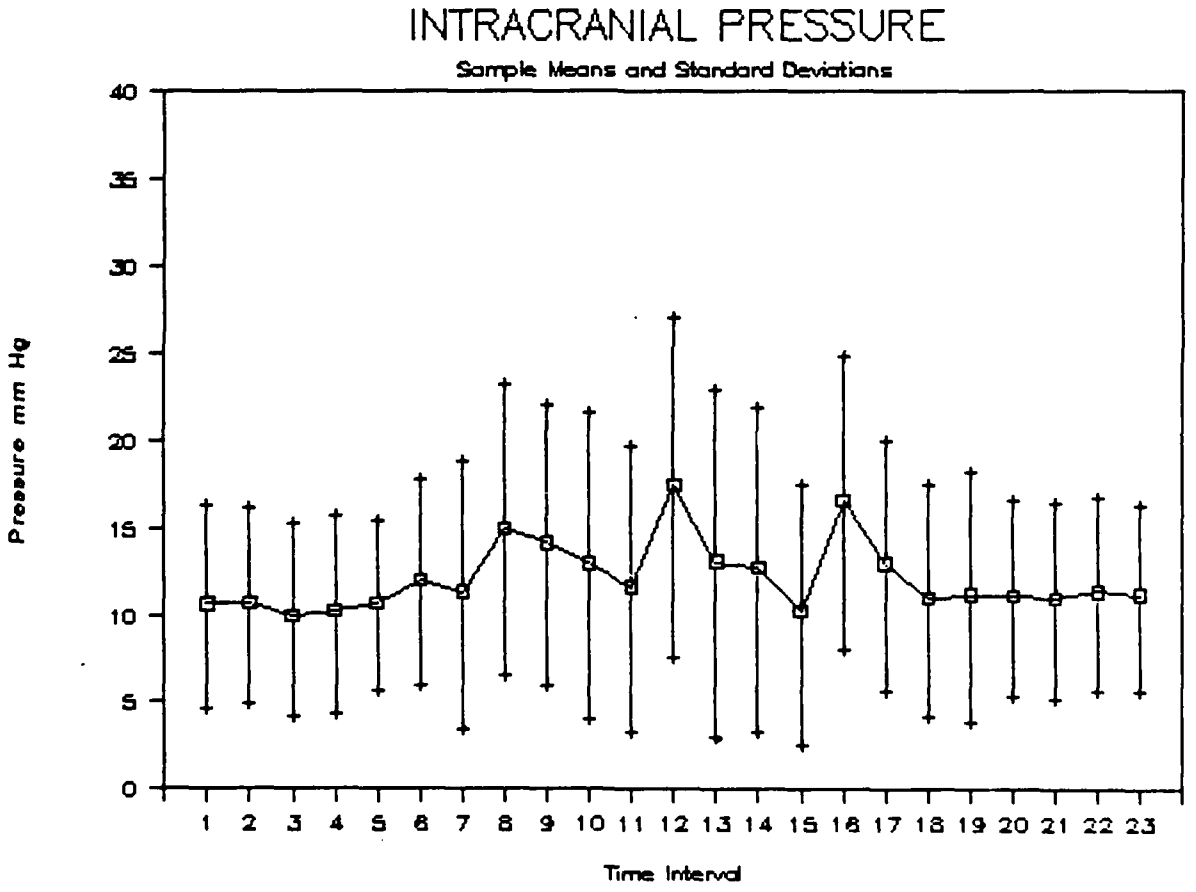
Nine subjects had GCS scores of 8T or below and four had GCS scores of 9T or 10T at the time of data collection. Sixty-two percent of subjects with spikes were male and 38 percent were female which closely patterns the demographics of the entire sample. The transient spikes do not appear to

correlate with any particular demographic variable.

In order to compute means for this variable, ANOVA's were done on various similar time intervals (baseline, suctioning, MH, and recovery phase) (Table 14).

Figure 7

Graphic Representation of
ICP Means and Standard Deviations
Across All Time Intervals



Baseline: Intervals 1-5

ETTS: Intervals 8, 12, & 16

MH: Intervals 6-7, 9-11, 13-15, & 17-18

Post Procedure: Intervals 19-23

Table 14

Repeated Measures ANOVA
Intracranial Pressure Time Intervals

Intervals	F	Sig. of F
1-5, 19-23	.55	.838
1-5 (M), 6-18, 19-23 (M)	3.60	.000*
MH 6-7, 9-11, 13-15, & 17-18	1.48	.158
Suctioning 8, 12, & 16	1.62	.212
1-5 (M), Suctioning (M), 19-23 (M)	6.87	.000*
MH 6-7, Suction 8	3.18	.053
MH 9-11, Suction 12	6.06	.001*
MH 13-15, Suction 16	4.46	.007*
Suction 16, MH 17-18	6.74	.003*

*significant ($p < .05$) (M)=mean of interval values

Because there was not a significant difference within time frames of baseline, MH, suctioning, and recovery period intracranial pressure values, means were computed for each of these time frames and an ANOVA was done to examine the overall effect of the procedure on ICP (Table 15).

Table 15

Repeated Measures ANOVA
Summary Table for Intracranial Pressure

Source	df	SS	MS	F	Sig. of F
Within Cells	57	1152.04	20.21		
TIME	3	417.83	139.28	6.89	.000

The means and standard deviations are presented graphically in Figure 8. A Tukey HSD Test was performed on the mean values to determine which intervals were significantly different. The intervals in which these significant differences occurred were baseline to ETTS, MH to ETTS, and ETTS to recovery period (Table 16).

Figure 8

Graphic Representation of
ICP Means and Standard Deviations
for Major Time Intervals

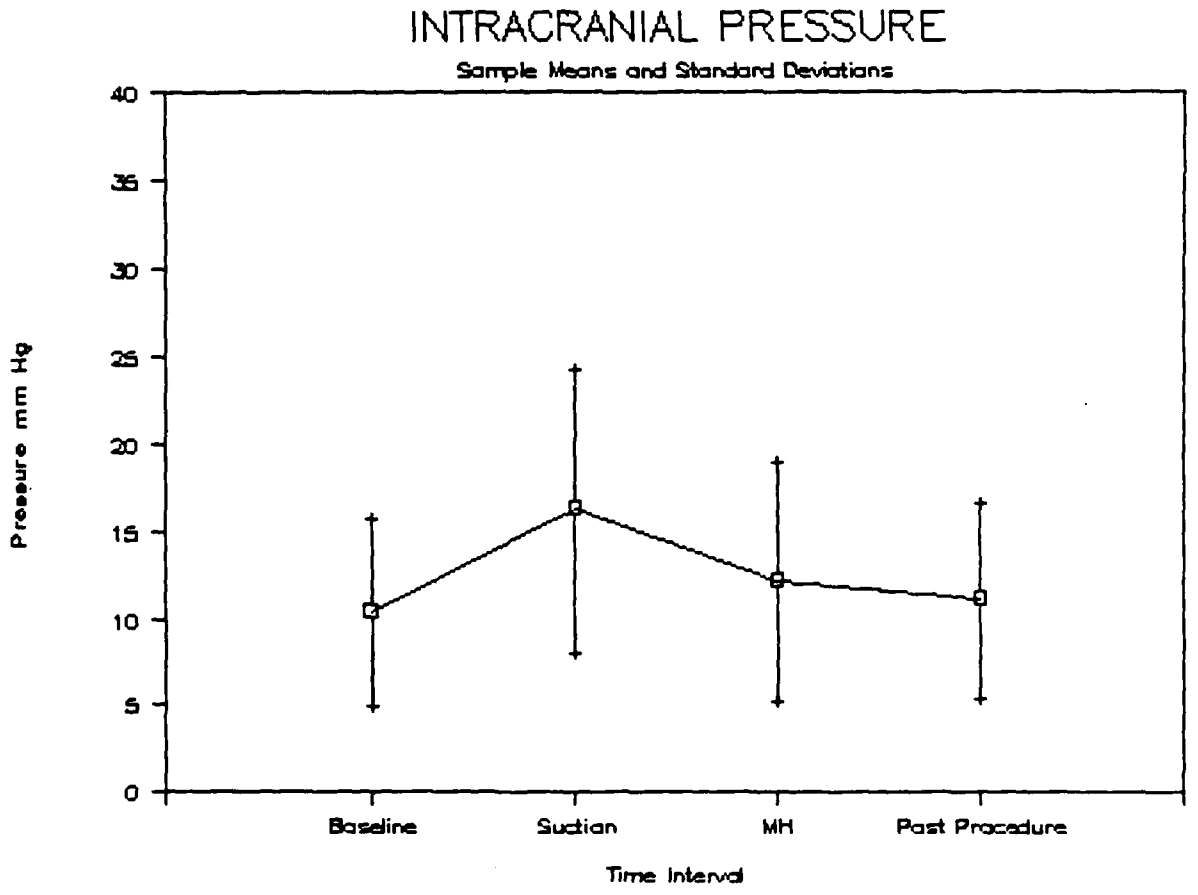


Table 16

Significant Time Interval Differences
Intracranial Pressure

	Base	Suction	MH	Recovery
Mean	10.40	16.31	12.19	11.15
Base		*		
Suction	*		*	*
MH		*		

*significant ($p < .05$)

Cerebral Perfusion Pressure

An analysis of variance for repeated measures was done to determine significant changes in cerebral perfusion pressure (CPP) during the ETTS/MH protocol. To determine this, an ANOVA was done across all 23 time intervals with a significant F value of 3.04 (p=.000) (Table 17). The means and standard deviations are presented graphically in Figure 9.

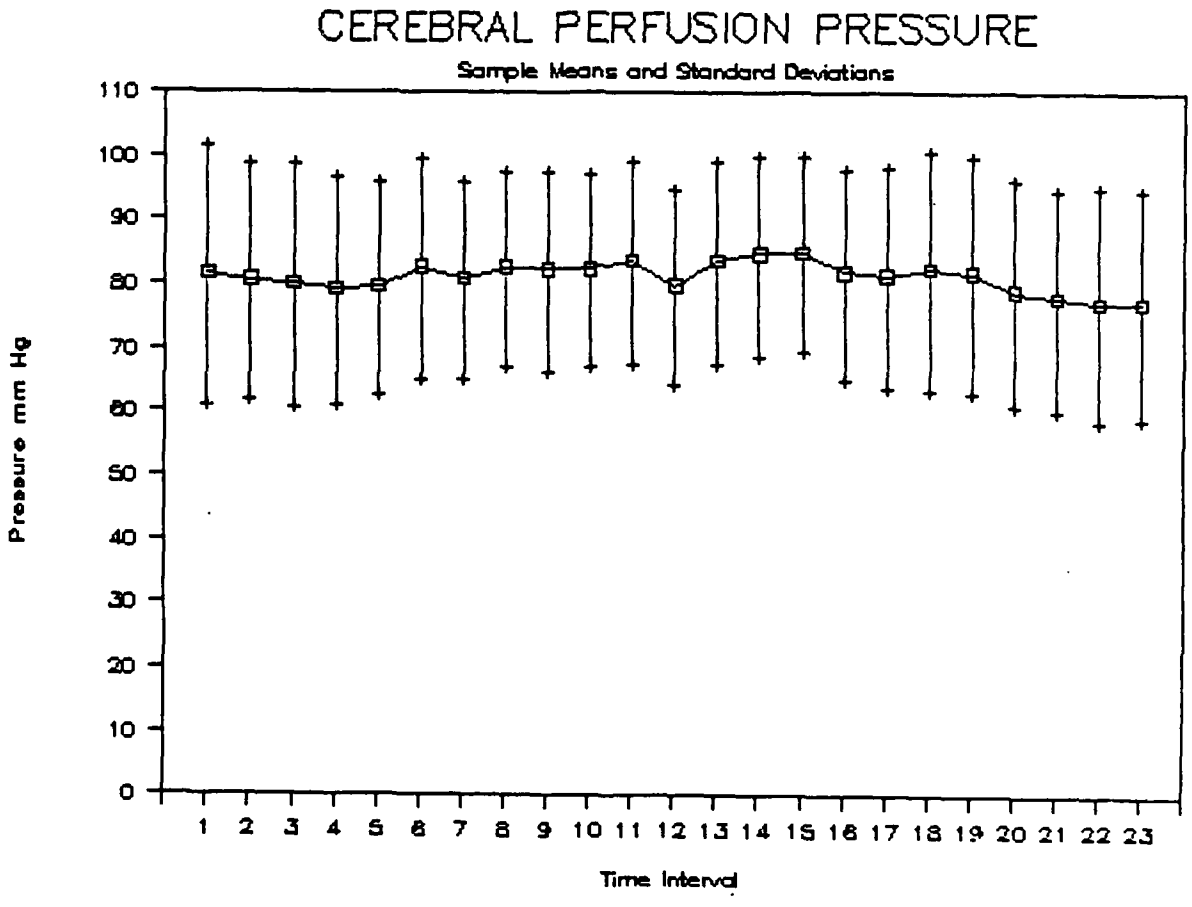
Table 17

Repeated Measures Analysis of Variance
Summary Table for CPP across 23 time intervals

Source	df	SS	MS	F	Sig. of F
Within Cells	418	13547.50	32.41		
TIME	22	2164.93	98.41	3.04	.000

Figure 9

Graphic Representation of
CPP Means and Standard Deviations
Across All Time Intervals



Baseline: Intervals 1-5

ETTS: Intervals 88, 12, & 16

MH: Intervals 6-7, 9-11, 13-15, & 17-18

Post Procedure: Intervals 19-23

In order to compute means for this variable, ANOVA's were done on various similar time intervals (baseline, suctioning, MH, and recovery phase) (Table 18).

Table 18

Repeated Measures ANOVA
Cerebral Perfusion Pressure Time Intervals

Intervals	F	Sig. of F
1-5	1.33	.266
19-23	4.95	.001*
1-5 (M), 19-23	2.46	.012*
1-5 (M), 6-18, 19-23	3.87	.000*
MH 6-7, 9-11, 13-15, & 17-18	1.56	.130
Suctioning 8, 12, & 16	1.35	.272
1-5 (M), Suctioning (M), 19-23	2.76	.007*
MH 6-7, Suction 8	.58	.565
MH 9-11, Suction 12	2.46	.072
MH 13-15, Suction 16	2.20	.098
Suction 16, MH 17-18	.29	.750

*significant ($p < .05$) (M)=mean of interval values

Because there was not a significant difference within time frames of baseline, MH, and suctioning, means were computed for each of these time frames and an ANOVA was done to examine the overall effect of the procedure on CPP. Although a significant difference existed within the recovery period time frame, a mean of those values was computed for this analysis (Table 19).

Table 19

Repeated Measures ANOVA
Summary Table for Cerebral Perfusion Pressure

Source	df	SS	MS	F	Sig. of F
Within Cells	57	1250.48	21.94		
TIME	3	202.79	67.60	3.08	.034

The means and stand d deviations are presented graphically in Figure 10. A Tukey HSD Test was performed on the mean values to determine which intervals were significantly different. The only interval in which a significant difference occurred was MH to recovery period (Table 20).

Table 20

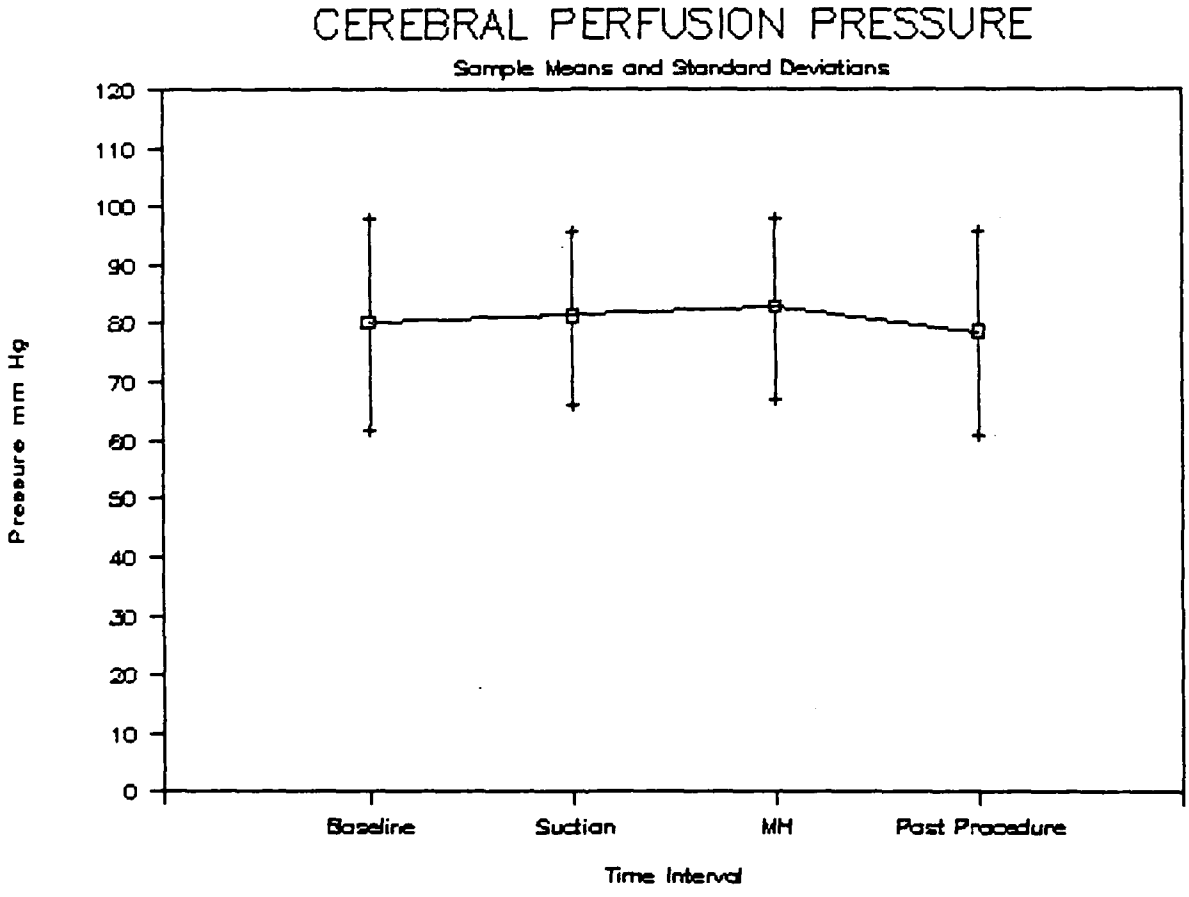
Significant Time Interval Differences
Cerebral Perfusion Pressure

	Base	Suction	MH	Recovery
Mean	80.20	81.45	82.91	78.59
Base				
Suction				
MH				*

*significant ($p < .05$)

Figure 10

Graphic Representation of
CPP Means and Standard Deviations
for Major Time Intervals



Oxygen Saturation

An analysis of variance for repeated measures was done to determine significant changes in oxygen saturation (O2 SAT) during the ETTS/MH protocol. To determine this, an ANOVA was done across all 23 time intervals. Statistical significance was not obtained with an F value of .96 (p=.514). Power was computed at .762 for this analysis. Note that only 19 subjects were included in this analysis.

Summary

Statistical analysis yielded a wealth of information which could be utilized to answer the proposed research question. The results reveal the effects of the ETTS/MH procedure on the cerebrovascular status of closed head injured patients as defined by sample demographics. The dependent variables of ICP, HR, MABP, and CPP were significantly effected by the ETTS/MH procedure. O2SAT was not significantly effected by the procedure.

The conclusions about the research question, based on these results and their interpretation will be presented in Chapter 5.

CHAPTER V

SUMMARY, CONCLUSIONS, IMPLICATIONS, AND RECOMMENDATIONS

Statement of Purpose

The purpose of this study has been to increase the body of knowledge regarding the effect of nursing activities on head injured patients. This study specifically examined the effects of endotracheal suctioning with manual hyperventilation (ETTS/MH) on the cerebrovascular status of severe closed head injured patients.

The purpose of this chapter is to summarize the research implemented and the results that were discovered. Conclusions drawn from the results of data analysis are presented. Implications for nursing practice based on the results of this study and recommendations for further research are discussed.

Summary

A major goal of the care of head injured individuals is the maintenance of adequate perfusion of the brain. This goal is realized, in part, by the prevention of intracranial hypertension and complications of immobility. Pulmonary complications of immobility can be diminished through regular pulmonary toilet which includes endotracheal suctioning for intubated patients. Endotracheal suctioning in conjunction with adequate ventilation and oxygenation minimizes hypoxemia and hypercapnia which have detrimental

effects on the cerebrovascular status of head injured patients. Serious questions as to the effects of routine nursing care procedures such as endotracheal tube suctioning on cerebrovascular status and brain perfusion have been raised.

The purpose of this study is to determine the effects of endotracheal tube suctioning with manual hyperventilation on the cerebrovascular status of severe closed head injured individuals. The theoretical framework of the study is based on the understanding that the normal brain maintains a system of regulation that matches cerebral blood supply to metabolic demand. The cerebral blood volume varies inversely with changes in cerebrovascular resistance. Injured brain tissue may not be able to compensate for increases in intracranial volume that occur secondary to changes in intracranial blood volume or the injury itself. The goal of therapeutic decision making must be to prevent significant and sustained increases in ICP to minimize destructive consequences of these conditions.

A quasi-experimental study was implemented utilizing a within-subject repeated measure design and a convenience sample of 20 mechanically ventilated patients with closed head trauma. All subjects were respiratory and cardiovascular stable at the time of study. Subjects received a standardized suctioning protocol which included manual hyperventilation of 30 to 45 seconds in duration and

three suction passes. Heart rate (HR), ICP, mean arterial blood pressure (MABP), cerebral perfusion pressure (CPP), and oxygen saturation (O2SAT) were monitored to assess cerebrovascular status. Subjects were continuously recorded using a four-cnannel recorder to quantify the dependant variables and provide a time dimension over which to assess the resultant values. Instrumentation included an ICP monitoring device, arterial pressure line, electrocardiography, Nellcor pulse oximetry unit, Mennen Series 480 bedside patient monitoring system, and a Mennen four-channel data recorder. Repeated measures Analysis of Variance (ANOVA) was computed for data analysis.

Results indicated that all dependent variables, except O2SAT were effected by the ETTS/MH procedure performed. Mean heart rate was highest during the ETTS and MH phases of the protocol with a value of 105.23 +/- 19.44 and 104.57 +/- 22.88 beats/minute respectively. Mean HR immediately returned to baseline values of 97.40 +/- 22.78 beats/minute within the first minute of the post-procedure period.

CPP remained adequate (> 50 mm Hg) in all subjects throughout the ETTS/MH procedure. Since cerebral autoregulation was proven to be intact, adequate cerebral blood flow throughout the treatment period was insured. Mean CPP increased from a baseline value of 80.20 +/- 18.26 mm Hg to 81.45 +/- 14.89 and 82.91 +/- 15.56 mm Hg respectively during the ETTS and MH sequences of the

protocol. Mean CPP dropped below baseline to 78.59 +/- 17.53 mm Hg during the post-procedure time frame, however it remained well above the minimal levels required to insure adequate cerebral blood flow.

Mean ICP was highest during suctioning 16.31 +/- 8.09 mm Hg. Mean ICP decreased to 12.19 +/- 6.84 mm Hg during the MH intervals immediately following each suctioning pass. Although mean ICP did not return to baseline level (10.40 +/- 5.37 mm Hg) after five minutes post-procedure, it did return to less than 1mm Hg above at 11.15 +/- 5.58 mm Hg. Thirteen subjects (65%) experienced transient ICP spikes of greater than 20mm Hg during one or more of the suctioning intervals (Appendix D). The spike pattern lasted from 3 to 6 seconds in duration and was not related to any specific demographic variables.

MABP became significantly elevated above baseline value of 90.60 +/- 19.36 mm Hg during the first MH time interval (94.55 +/- 18.96 mm Hg). To enhance CPP in the presence of elevated ICP's, MABP remained elevated during the entire ETTS/MH procedure reaching the highest levels during each of the suctioning intervals with a mean of 97.80 +/- 17.44 mm Hg. The third suctioning pass produced the highest pressure (98.50 +/- 18.67 mm Hg). MABP values dropped below baseline to 89.74 +/- 17.85 during the post-procedure time frame.

O2SAT had negligible changes during the ETTS/MH procedure and had no statistical or clinical effect on the cerebrovascular status of the subjects.

Conclusions

It is concluded that endotracheal suctioning of 15 seconds or less in duration with manual hyperventilation of 45 seconds can be performed without detriment to the cerebrovascular status in adult patients who have sustained severe closed head injury and have baseline ICP's of 0 to 20 mm Hg, providing CPP is maintained at a level of 50 mm Hg or greater.

CPP remained constant in the presence of ICP and MABP changes during the protocol which suggests proper functioning of the cerebral autoregulatory processes. Because rises in MABP were not accompanied by an increase in ICP, it is assumed that autoregulation is intact (See Appendix I). If autoregulation was not intact, cerebral resistance vessels would not have constricted in response to MABP elevations which would have increased cerebral blood flow and corresponding increases in ICP. Although CPP drops during the post-procedure period, this is not clinically significant unless it falls near the lower limit of 50 mm Hg.

MABP rose during the ETTS/MH procedure. This finding is supported by the rationale that ICP elevations stimulate the cerebral ischemic response subsequently activating the

sympathetic nerves to increase HR and MABP (Miller & Sullivan, 1978). Other nursing studies support the findings of elevations in MABP coinciding with rises in ICP (Parsons, Peard, & Page, 1985; Parsons & Shogan, 1984; Parsons & Wilson, 1984). Other factors which could contribute to the observed rise in MABP during suctioning intervals are the cough reflex or the noxious stimuli of suctioning precipitating an increase in arousal and release of catecholamines which contribute to MABP elevations (Lundberg, 1960; Snyder, 1983).

The MABP returned to baseline level during the first minute post-procedure. It continued to drop below baseline levels at five minutes post-procedure. The clinical significance of the observed changes in MABP lies in the relationship of MABP to CPP. In the presence of a rapidly stabilizing ICP, a fall in MABP will most likely result in a diminished CPP. This conclusion is supported by studies with similar designs (Parsons & Shogan, 1984; Parsons, Peard, & Page, 1985) in which the same relationship between MABP and CPP exist. This is the rationale for providing a rest period between suctioning and other patient and nursing activities in which the cardiovascular and cerebrovascular parameters may stabilize.

Examination of the mean ICP values during the ETTS and MH time intervals reveals that the ICP was highest during the 15 second suctioning intervals. These values

immediately returned to levels which were not significantly different from baseline during the MH sequence immediately following each suction pass. Increased pressure in the cranial cavity is a normal but transient event of such everyday activities as coughing; however, when transient increases in ICP are prolonged, adequate perfusion becomes a concern (Mitchell, 1986). ICP monitoring during the ETTS/MH procedure allows for suctioning to be aborted if elevation in ICP exceeds clinically acceptable limits thereby preventing cerebrovascular compromise.

The transient elevation in ICP during suctioning can be attributed to the fact that 65% (13) of the subjects experienced transient ICP spikes of greater than 20mm Hg over baseline. The spike patterns were transient in nature and lasted from 3 to 6 seconds in duration. There were no consistent demographic patterns which would explain the difference between patients who had spikes and the rest of the sample. This suggests that suctioning technique, irritation by the endotracheal tube, or amount of manipulation of the endotracheal tube during the suctioning process could have impacted these patterns. Sedation and patient positioning such as proper head support to promote venous return and raising the head of the bed during the suctioning procedure are essential considerations to promote lower baseline ICP's that would enable patients to better tolerate the suctioning procedure.

Heart rate became elevated during the first MH sequence and remained elevated throughout the three minute time interval during ETTS and MH. It immediately returned to baseline in the post-procedure time period. The rationale for this increase can be attributed to those factors previously cited for rises in MABP, including activation of the central ischemic response and catecholamine activation. These findings are consistent with other studies which report similar elevations in HR related to increased ICP (Grof & Rossi, 1978; Parsons & Shogan, 1984, McQuillan, 1986). An increase in HR is expected during a procedure such as suctioning which stimulates coughing and changes in intrathoracic pressures (MacKenzie et. al., 1981; Tsementzis, Harris, & Loizou, 1982). A study of suctioning and hyperventilation (Parsons & Shogan, 1984) reported similar patterns in HR response as those found in this study.

An adequate cardiac output is essential for competent cerebral blood flow (Gaab & Heissler, 1984). A heart rate of less than 40 beats per minute (bpm) or greater that 160 bpm significantly diminishes cardiac output (Little, 1981). The heart rate stayed well within these parameters thereby insuring adequate cardiac output to support cerebral blood flow.

Oxygen Saturation was not significantly effected by the ETTS/MH procedure. No other studies have reported the

effects of this procedure on O2SAT. The lack of significant variation during the ETTS/MH procedure is not surprising given all subjects in this study had a stable respiratory status, were mechanically ventilated, and well oxygenated prior to the start of the protocol. Studies have shown that decreases in PaO2 occur during or immediately following endotracheal tube suctioning (Adlkofer & Powaser, 1978; Chulay & Graeber, 1988; Erhardt, Hoffman, & Loveland, 1981, Jung & Newman, 1982; Skelley, Deeren, & Powaser, 1980). Because hypoxemia has a strong cerebral vasodilatory effect, and therefore influences intracranial pressure and cerebral perfusion, it is essential to have a physiological measurement tool which will be sensitive to these changes. Based on the findings with this patient population, pulse oximetry oxygen saturation measurement does not appear to be a sufficient measure.

It is concluded that the benefits of prevention of pulmonary complications of immobility utilizing endotracheal suctioning of 15 seconds or less in duration with manual hyperventilation of 45 seconds, can be obtained without detriment to the cerebrovascular status in adult patients who have sustained intracerebral trauma.

Limitations

During the course of implementation of this study, several limitations were identified which were not anticipated during its formulation.

Pulse oximetry was not found to be sensitive enough to changes in oxygenation during the ETTS/MH procedure. There was very little alteration in this variable which is attributed to the fact that the subjects were all well oxygenated and had respiratory stability prior to the start of the protocol.

The study did not measure PaO2 or PaCO2 during the protocol. As a result, the effect of the ETTS/MH procedure on these sensitive metabolic parameters could not be determined. One can only speculate on the effect of too much hyperventilation on the cerebrovascular status in this patient population.

Implications for Nursing Practice

The results of this study provide nurses with knowledge of the physiologic changes that occur in cerebrovascular status during endotracheal suctioning with manual hyperventilation as it is currently being employed. These findings can only be applied to patients that fall into similar demographic parameters.

This study suggests that suctioning with manual hyperventilation can safely be performed upon severe closed head injured persons whose resting mean intracranial pressure is less than 20 mm Hg without compromising cerebral blood flow. Cerebral perfusion pressures should be kept above 50 mm Hg during this intervention.

The results of this study identify a cumulative effect

of the suctioning procedure on cerebrovascular parameters, especially CPP, therefore head injured patients should be given periods of rest between suctioning and other treatments or patient and nursing activities. Recognizing the extensive variation in type and severity of head injuries and the unpredictable patient response to these cerebral insults, it is important that meticulous monitoring of ICP and MABP values to determine adequacy of CPP be implemented throughout this procedure.

The effects of transient changes in cerebrovascular status on long-term recovery of head injured patients is not clear. Patients experiencing ICP spikes during the suctioning procedure should have periodic recordings of the spike patterns to determine and document amplitude, frequency and duration. Significant increases in ICP were recorded in this sample when spiking occurred and they represent potential cerebrovascular compromise if frequency and duration are not minimized.

Parsons and Shogan (1984) suggest that multiple manual hyperventilations after the third and subsequent ETTS's should be extended to a longer time interval (from 20 to 30 seconds), perhaps 60 seconds, in order that the physiologic measurements of MABP, ICP, CPP, and HR more closely approach the baseline levels. The results of this study do not support this recommendation. The longest manual hyperventilation sequence of 45 seconds in this study was

sufficient in this sample to allow these physiologic measures to return close to baseline levels. The concern of this author is that too much hyperventilation may lead to high PaO2 and low PaCO2 levels which, conceptually, could lead to cerebral ischemia, especially in patients being maintained on mechanical hyperventilation for ICP control. Hyperventilation duration should therefore be minimized to the shortest time duration to prevent intracranial hypertension, thereby insuring maintenance of cerebral blood flow.

Recommendations for Further Research

Since only two other studies exist which systematically describe the cerebrovascular changes during suctioning and manual hyperventilation of adult head injured patients, replication of this study is essential. Study of a larger number of individuals will allow greater generalizability to the head injured population as well as identifying the wide variety of individualized responses to the suctioning procedure.

This study only included patients with closed head injuries. Extending this study to include patients with other causes for increased ICP such as CVA's, encephalitis and open head injuries is proposed to further determine effects of this procedure on cerebrovascular parameters in the presence of increased pressures.

The sample in this study included only adult patients.

The systematic study of the effect of the ETTS/MH procedure on other populations such as children or the elderly is another area for future study.

This study did not measure PaCO2 or PaO2 during the protocol. As a result, the effect of the ETTS/MH procedure on these sensitive metabolic parameters could not be determined. A study designed to determine the optimal hyperventilation method which would prevent hypoxemic and hypercapnic episodes still be sufficient to maintain adequate cerebral blood flow is another area for further research.

Other researchers are examining the effects of suctioning with hyperinflation on cerebrovascular status. A study using the same sample and designed to compare the hyperinflation and hyperventilation effects on cerebrovascular status is proposed.

The response of patients with elevations in baseline ICP greater than 20 mm Hg to the ETTS/MH procedure has not been studied. These patients are not exempt from the complications of immobility and therefore require pulmonary toilet measures to assist in prevention of pulmonary complications. A study designed to address the specific response of this population to these measures is indicated so that respiratory care activities can be performed with the least detrimental effect.

Further research regarding the effects of respiratory

care on cerebrovascular status as well as refinement of nursing practice to minimize these effects is indicated. It is hoped that the results of this study will encourage other researchers to investigate the myriad of unanswered questions pertaining to the effectiveness of nursing interventions in the care of neurologically compromised patients.

APPENDIX A

CALIBRATION OF MENNEN 4 CHANNEL DATA RECORDER

Standard Protocol

Channel 1: Heart Rate
Channel 2: SaO2
Channel 3: Blood Pressure (BP1)
Channel 4: ICP (BP2)

Paper Speed: 5mm/sec

Attaching Recorder to Monitor System

1. Attach 4 cables to back of monitor by matching numbers on cable to plug sites.
2. Plug cables 1,3, and 4 into front of data recorder by matching numbers on cable to jack sites.
3. Plug cable from pulse oximeter into jack #2 on front of data recorder.

Calibrating Pressure Channels

1. Channel 3: Blood Pressure
   a. Make sure BP-1 (A-Line) is in Channel 3.
   b. Set DISPLAY to BALANCE.
   c. Set MODE to STANDBY.
   d. Set RANGE to 300.
   e. Open transducer to air (Off to patient).
   f. Press AUTO ZERO (Should read 00 +/- 1).
   g. Set MODE to CALIBRATE (0-300 should alternate on display).
   h. Adjust SIZE button on recorder to calibrate top of pattern with top of Channel 3 strip.
   i. Adjust POSITION button on recorder to calibrate bottom of pattern with bottom of Channel 3 strip.
   j. Set MODE to STANDBY.
   k. Open transducer to patient (Off to air).
   l. Wave form will appear on recorder.
   m. Set DISPLAY to S/D.
   n. Mark "0-300" on paper channel 3 and put values monitor is reading as a point of reference.

2. Channel 4: ICP
   a. Make sure BP-2 (ICP) is in Channel 4.
   b. Set DISPLAY to BALANCE.
   c. Set MODE to STANDBY.
   d. Set RANGE to 50.
   e. Open ICP transducer to air (Off to patient) using sterile technique with mask in place. Ask nurse if they want to do this or let researcher accomplish.
   f. Press AUTO ZERO (Should read 00+/- 1).
   g. Set MODE to CALIBRATE (0-50 should alternate on display).
   h. Adjust SIZE button on recorder to calibrate top of pattern with top of Channel 4 strip.
   i. Adjust POSITION button on recorder to calibrate bottom of pattern with bottom of Channel 4 strip.
   j. Set MODE to STANDBY.
   k. Open transducer to patient (Off to air) and redress immediately using sterile technique.
   l. Wave form will appear on recorder.
   m. Set DISPLAY to S/D.
   n. Mark "0-50" on paper channel 4 and put values monitor is reading as a point of reference.

3. Channel 1: Heart Rate
   a. To minimize artifact, plug cable from ECG IN/OUT jack on back of pulse oximeter into QRS SYNCH jack on bottom front of monitor system.
   b. Adjust SIZE and POSITION button on recorder to insure rhythm pattern is displayed on Channel 1 strip.

4. Channel 2: SaO2

   a. Attach Nellcor Pulse Oximetry unit into Channel 2 jack on front of data recorder.
   b. Make sure pulse oximeter is OFF.
   c. Press 0 on back of oximeter and adjust SIZE button on recorder to calibrate bottom of pattern with bottom of Channel 2 strip.
   d. Press FULL on back of oximeter and adjust POSITION button on recorder to calibrate top of pattern with top of Channel 2 strip.

APPENDIX B

The Glascow Coma Scale

Category of Response	Response	Score
Eye Opening	Spontaneously	4
	To speech	3
	To pain (but not speech)	2
	None	1
Best Verbal Response	Oriented	5
	Confused conversation	4
	Inappropriate words	3
	Incomprehensible sounds	2
	Mute	1

If intubated and unable to test this category, T appears next to the score.

Best Motor Response	Obeys commands	6
	Localizes to pain	5
	Semipurposeful	4
	Flexor response	3
	Extensor response	2
	Flaccid	1

(Kenner, Guzetta, and Dossey, 1985, Critical Care Nursing: Body-Mind-Spirit)

APPENDIX C

The Effects of Selected Activities on ICP in Closed Head Injury

SUBJECT ID #_____

AGE____ SEX____ RACE____ DATE

INJURED_____

Date Time

ADMISSION DATA

BLOOD ETOH

GCS

PRIMARY DIAGNOSIS

INJURIES:	MILD	MODERATE	SEVERE
HEAD INJURY			
SPINAL CORD INJURY			
SKELETAL			
CARDIAC			
VASCULAR			
RESPIRATORY			
RENAL			
GASTROINTESTINAL			
REPRODUCTIVE			
OTHER			

DATA COLLECTION SHEET

ID PROTOCOL

DAYS SINCE INJURY CURRENT GCS

ICP (LAST 8 HRS) (RANGE) (MEAN)

DATE ICP MONITORING INITIATED TYPE:

MOST RECENT ABG (date and Time):

pO2 pCO2 pH SO2

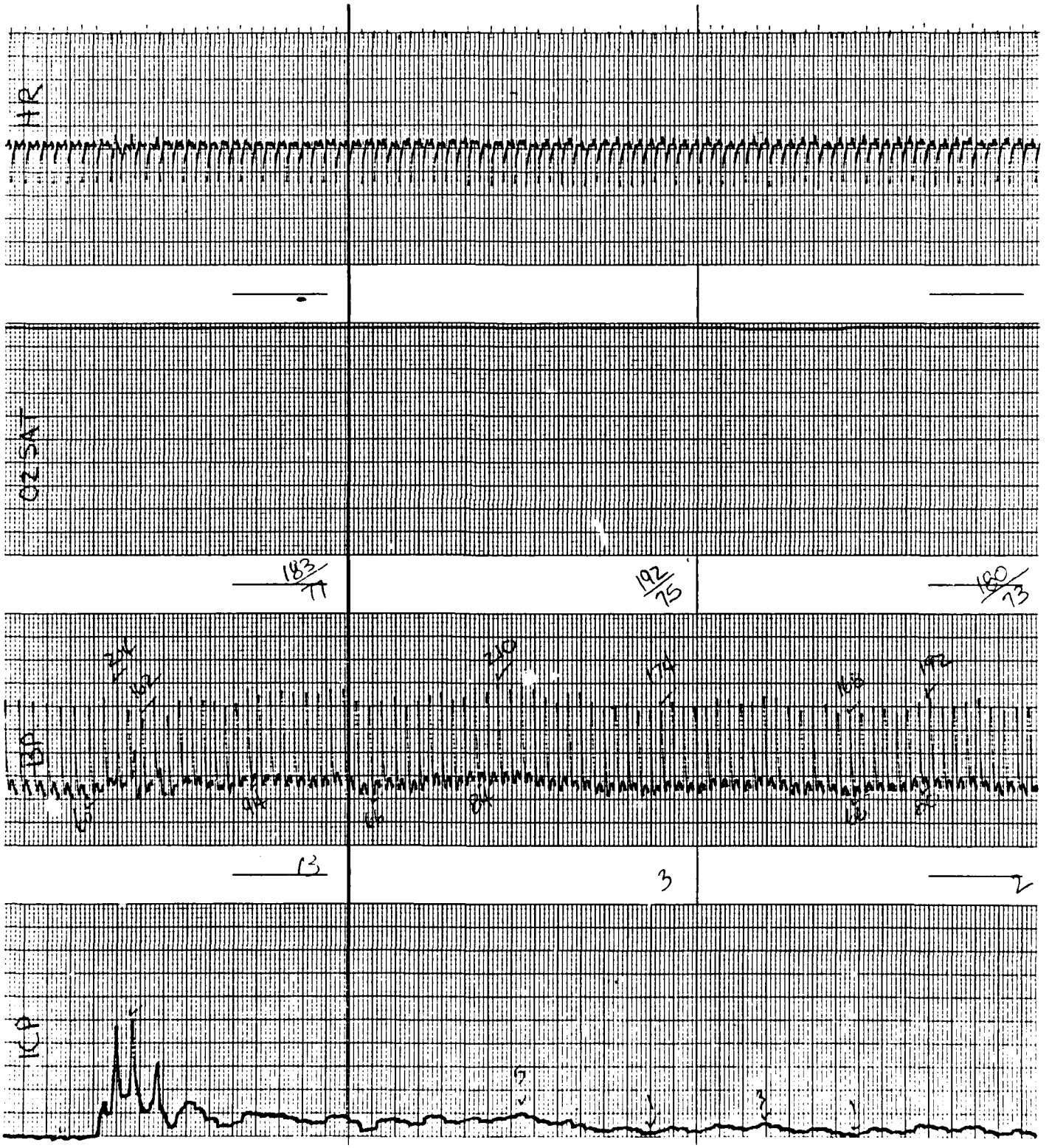
FIO2 PEEP Temperature

CURRENT DRUG REGIMEN

DRUG	DOSE	TIME OF LAST DOSE
ANTICONVULSANT		
BARBITUATE		
NARCOTIC		
PARALYTIC		
STEROID		
DIURETIC		
INOTROPE		
VASODILATOR		

APPENDIX D

Mennen Four Channel Data Recorder Paper



APPENDIX E

Sample Means and Standard Deviations Across Time Intervals Heart Rate

Time Interval		Mean	Standard Deviation
1	(Baseline)	97.20	22.63
2		96.20	22.53
3		96.40	22.66
4		97.40	23.83
5		99.80	22.79
6	(MH)	102.80	24.28
7		103.20	24.33
8	(Suction)	106.50	21.41
9	(MH)	105.60	20.98
10		103.20	23.91
11		102.20	24.91
12	(Suction)	104.20	20.90
13	(MH)	104.60	22.26
14		105.20	24.00
15		104.20	24.74
16	(Suction)	105.00	19.67
17	(MH)	107.70	23.98
18		107.00	22.32
19	(Post Procedure)	99.40	23.08
20		98.40	22.64
21		98.00	22.97
22		99.60	24.44
23		99.20	23.98

APPENDIX F

Sample Means and Standard Deviations Across Time Intervals Mean Arterial Blood Pressure

Time Interval	Mean	Standard Deviation
1 (Baseline)	92.20	20.86
2	91.25	19.76
3	89.90	20.03
4	89.35	19.25
5	90.30	17.95
6 (MH)	94.55	18.96
7	92.05	18.84
8 (Suction)	97.60	18.38
9 (MH)	96.50	18.15
10	95.45	17.97
11	95.35	18.71
12 (Suction)	97.30	17.37
13 (MH)	96.65	19.59
14	97.60	20.49
15	95.20	17.22
16 (Suction)	98.50	18.67
17 (MH)	94.35	17.56
18	93.50	18.25
19	92.95	17.58
20 (Post Procedure)	90.10	17.59
21	88.90	17.89
22	88.45	18.90
23	88.30	18.48

APPENDIX G

Sample Means and Standard Deviations Across Time Intervals Intracranial Pressure

Time Interval	Mean	Standard Deviation
1 (Baseline)	10.60	5.86
2	10.65	5.61
3	9.90	5.53
4	10.20	5.68
5	10.65	4.83
6 (MH)	12.00	5.94
7	11.30	7.65
8 (Suction)	14.95	8.33
9 (MH)	14.15	8.06
10	13.00	8.77
11	11.60	8.17
12 (Suction)	17.45	9.81
13 (MH)	13.05	9.98
14	12.75	9.35
15	10.20	7.45
16 (Suction)	16.55	8.38
17 (MH)	12.95	7.14
18	10.95	6.65
19 (Post Procedure)	11.15	7.19
20	11.10	5.60
21	11.00	5.63
22	11.35	5.55
23	11.15	5.29

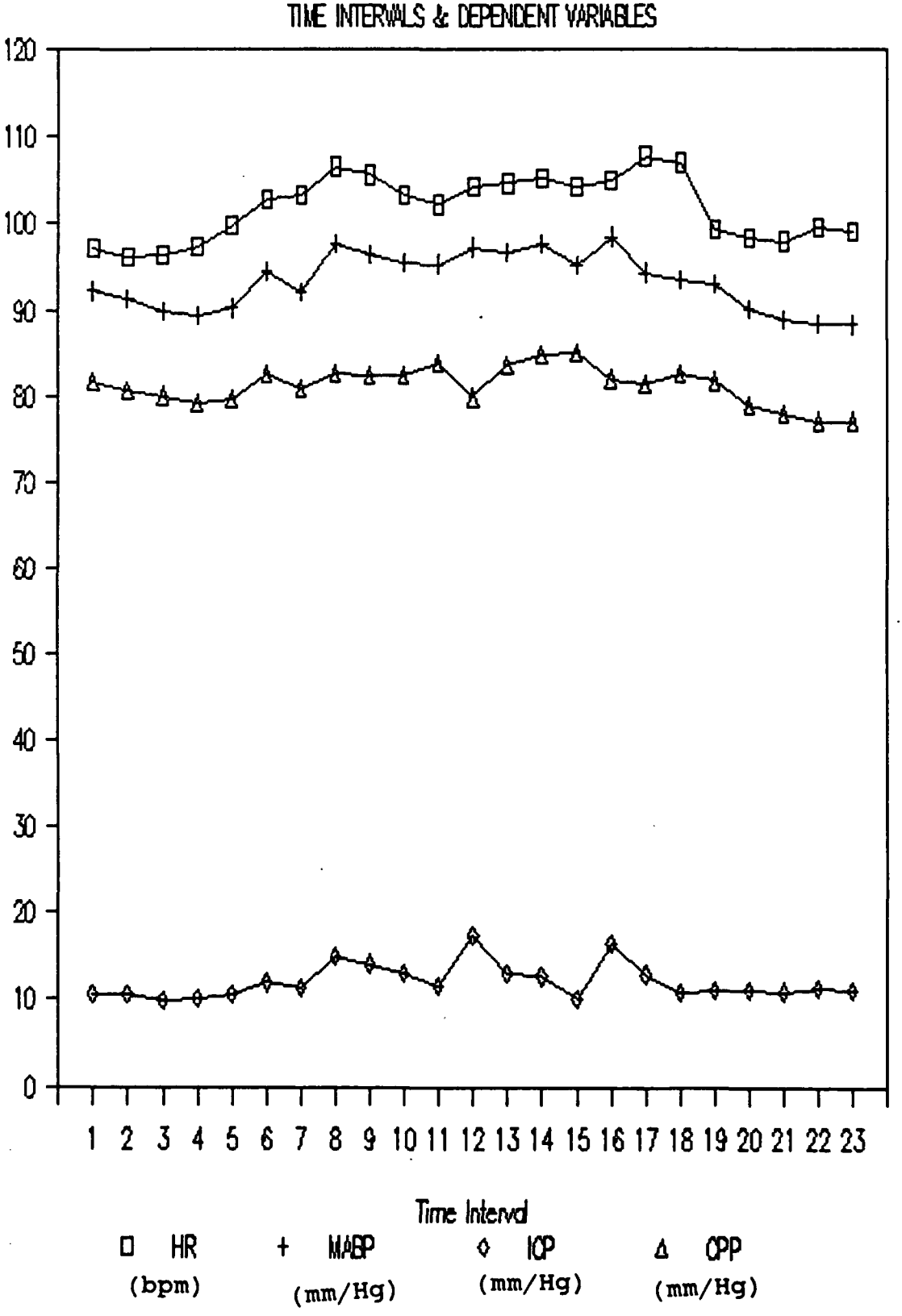
APPENDIX H

Sample Means and Standard Deviations Across Time Intervals Cerebral Perfusion Pressure

Time Interval	Mean	Standard Deviation
1 (Baseline)	81.60	20.42
2	80.60	18.62
3	80.00	18.92
4	79.15	17.93
5	79.65	16.64
6 (MH)	82.55	17.17
7	80.80	15.56
8 (Suction)	82.60	15.27
9 (MH)	82.40	15.63
10	82.40	15.07
11	83.75	15.82
12 (Suction)	79.80	15.04
13 (MH)	83.60	15.80
14	84.75	15.73
15	85.00	15.12
16 (Suction)	81.95	16.42
17 (MH)	81.40	17.26
18	82.50	18.70
19 (Post Procedure)	81.80	18.50
20	79.00	17.64
21	77.90	17.20
22	77.10	18.25
23	77.150	17.80

APPENDIX I

Graphic Means Across all Time Intervals and Dependent Variables



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